CARDIAC EMERGENCIES AND HEART FAILURE

Prevention and Treatment

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Preface

THE prompt diagnosis of cardiovascular emergencies and their proper treatment are among the most common problems confronting the general practitioner. This monograph has been written in the hope that it will be of value to him as he encounters these acute conditions. It is not intended to be a book on Cardiology, Radiology or Electrocardiography; consequently it does not cover these fields as thoroughly as do the standard textbooks. Physiology and pathology are discussed when a fuller understanding of them is considered necessary for the intelligent handling of cardiac patients. The drugs that have been found valuable are stressed; other drugs in current use are discussed. Case histories are presented to illustrate specific conditions and the use of the newer drugs. Throughout the book the importance of preventing cardiac emergencies by adequate interval therapy is emphasized.

We have included sections on surgical cardiac emergencies, on cardiac resuscitation and on certain acute conditions that are not commonly encountered, i.e., dissecting aneu

rysm hypertensive crises secondary to pheochromocytoma and the acute episode resulting from a "ball valve" thrombus in the hope that when these emergencies are seen they may be recognized and treated properly

The section on congestive heart failure and its complications is brief but we have included all of the information that we believe is necessary for the treatment of this condition

We have surveyed the literature but have drawn freely from our own cases and experience as well as from material previously published by one of us (*A M M*) Portions of this monograph have been presented in several broadcasts sponsored by the New York Academy of Medicine over Station WNYC (FM)

We wish to thank Dr Samuel Kahn and Dr Phillip Samet for editorial assistance We are indebted to members of the cardiographic department of the Mount Sinai Hospital for valuable suggestions

We hope that the great number of physicians who are called upon to diagnose and treat the diseases that we discuss will benefit from this work

New York N Y

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CARDIAC EMERGENCIES AND HEART FAILURE

Introduction

Due to the tremendous advances in the prevention and treatment of many diseases, and the increased span of life, cardiovascular disease now greatly outnumbers all others and causes more than half the deaths after the age of fifty Acute heart conditions require emergency treatment more than do those of any other organ making the term ' acute heart ' even more appropriate than the expression ' acute abdomen " The prompt treatment of acute cardiac disease by the application of well established rules and the utilization of recent advances in the field of cardiology, will certainly save many lives and hasten recovery in others Since treatment must be immediate, it is incumbent upon the physician to know how to diagnose and treat cardiac emergencies, and to have the essential drugs readily available This requires the careful selection and constant replenishment of medical supplies for it is very disheartening to make the correct diagnosis of a cardiac emergency, and not have the suitable medication available The following materials should always be at hand

1 Aminophyllin 2 cc ampules = 0 48 gm for i m , 10 cc ampules = 24 gm for i v use or 5 gm suppositories

2 Atropin Sulfate 75 mg (gr 1 100) tablets for intravenous or oral use

3 Caffeine Sodium Benzoate 2 cc ampules (5 gm) for i m use

4 Cedilanid 4 cc ampules 8 mg (Lanatocide C)
5 Demerol 100 mg tablets
6 Digoxin 25 mg tablets, 1 cc = 5 mg ampules (must be diluted in 10 cc saline for i v use)
7 Dilaudid hypo tablets 2 mg (gr 1/32) or ampules 1 cc = 2 mg (gr 1/32)
8 Epinephrine 1 1000—(1 cc)
9 Magnesium Sulfate 10 cc of 20 per cent solution ampules
10 Mercurial Diuretic (Mercuhydrin or Thiomerin) 1 cc or 2 cc
11 Morphine Sulfate 8 mg (1 8 gr) or 16 mg (1/4 gr) tablets for i m or i v use
12 Needles for phlebotomy
13 Neosynephrin (ampules for i v use) 1 cc = 10 mg
14 Nitroglycerin 30 mg (gr 1 200) or 40 mg (gr 1 150) hypodermic tablets
15 Potassium Chloride 20 cc ampules = 3 7 gm tablets 3 gm (5 gr)
16 Pronestyl (Procaine Amide) 250 mg capsules 10 cc ampules containing 100 mg per cc
17 Quinidine 2 gm (3 gr) and 3 gm (5 gr) tablets 2 gm (1 cc) ampules for i m use
18 Sodium Amytal 06 gm (gr 1) and 2 gm (gr 3) capsules for oral use 5 gm of powder in ampules for parenteral use
19 Sterile water for dilution of tablets
20 Strophanthin (K Strophanthin) 1 cc = 65 mg or Ouabain (G Strophanthin) 1 cc = 25 mg
21 Syrup of Ipecac
22 Tourniquets

It is surprising to find that a great number of physicians do not carry even the few items listed above, and are forced to use other less satisfactory preparations or call the "corner drug store' for supplies

Chapter

1

ARRHYTHMIAS

A CHANGE in the rate or rhythm of the heart is a common cause of acute distress in patients with and without cardiac disease Even in a patient with a normal heart an arrhythmia may produce many symptoms such as pounding, fright, precordial aching or severe pain,[71] vertigo syncope, vomiting, collapse, and rarely congestive heart failure [307] The most severe circulatory derangements occur following the onset of ectopic arrhythmias with rapid rate, *i e*, the paroxysmal tachycardias

PAROXYSMAL TACHYCARDIA

The paroxysmal tachycardias include auricular flutter, and fibrillation, auricular, nodal and ventricular tachycardia and ventricular fibrillation These tachycardias often remit spontaneously within several hours and cause no disturbance, but they may produce severe symptoms and marked effects on circulatory dynamics A change from a normal heart rate of 70 to 90 to one of 120 to 130 usually increases minute volume output for despite the fact that ventricular filling and stroke volume may be decreased, the product of heart rate and stroke output is increased When the heart rate is between 120 to 130 and 180, however, no further increase in minute output occurs With further increases in cardiac rate, total minute volume is actually reduced [114] This may result in severe cerebral anoxia, fainting, or convulsions

J C, a thirty two year old patient studied by us, was a tense person and a factory manager, who fainted on several occasions He gave a long history of discomfort whenever he wore a tie and his pulse slowed moderately on carotid sinus pressure The fainting was therefore considered to be the result of carotid sinus sensitivity Close questioning however revealed that the syncope was sometimes preceded by palpitation The patient was later seen during an episode of nodal tachycardia This was doubtless the major cause of his fainting although the carotid sinus may also have played a role

If the tachycardia is allowed to persist it may result in a further decrease in cardiac output shock and/or cardiac failure [247] A picture of coronary insufficiency with chest pain and RS T and T wave changes in the electrocardiogram may also occur if the tachycardia continues We have seen many such patients one of whom was a sixty five year old woman with carcinoma of the left lung and hemo thorax who was receiving nitrogen mustard therapy On the fifth day after therapy she suddenly experienced precordial pain and palpitation and went into shock Her pulse rate was 180 A cardiogram revealed a 2 1 auricular flutter with RS T depressions in all leads While the cardiogram was being taken the rhythm reverted to normal but the RS T depressions persisted for several minutes before disappearing In many instances these RS T depressions persist for hours or days

The *supra ventricular tachycardias i e* auricular or nodal tachycardia and auricular flutter and fibrillation, may occur in both normal and abnormal hearts [27, 145] and may be precipitated by emotional disturbances [72] Occasionally, they are the only indication of hyperthyroidism although a recent study has shown that the basal metabolic rate is within normal limits in over 85 per cent of all cases of supra ventricular tachycardia [143] Most commonly, these arrhyth

mias occur in patients who have rheumatic or arteriosclerotic heart disease or in those whose hearts are normal Ventricular tachycardia on the other hand is almost always associated with organic heart disease particularly coronary occlusion [15] and is of more serious import

In many normal persons with paroxysmal tachycardia therapy may be withheld for several hours since the tachycardia often remits spontaneously Individuals with normal hearts may be able to tolerate rapid heart rates for weeks or months without exhibiting any detectable alterations in cardiac function [89 229] Some patients however are emotionally disturbed and may experience precordial discomfort as a result of prolonged rapid beating of the heart in such patients treatment should be started early

Certain general measures are of value in the prevention and treatment of all tachycardias namely rest sedation and the avoidance of tobacco alcohol caffeine and emotional upsets A twenty eight year-old physician recently observed developed auricular fibrillation whenever he took one drink and smoked one cigarette simultaneously He was finally persuaded to stop drinking and smoking and experienced no further attacks Occasionally the ingestion of small amounts of alcohol will actually prevent the recurrence of tachycardias although this is not common Eating fatty foods or large meals on rare occasions produces auricular arryhthmias in patients with otherwise normal hearts We have observed a patient who suffered a bout of auricular tachycardia each time he ate a frankfurter or a large dish of ice cream The avoidance of these foods completely eliminated the attacks

A correct diagnosis is important before specific therapy for paroxysmal tachycardia can be instituted It should be remembered that any of the tachycardias may be transitory and it is best to wait several hours before beginning drug therapy except in cases where the patient is in shock or cardiac failure

AURICULAR FIBRILLATION

Auricular fibrillation is the most common serious arrhythmia and occurs most frequently in patients with rheumatic and arteriosclerotic heart disease[3] It may be seen in cases of Graves disease[50] following operative procedures[85] in pneumonia and in other febrile illnesses Both the paroxysmal and chronic types are seen occasionally in the absence of organic heart disease[4, 9, 98]

Auricular fibrillation has been attributed by some observers to rapid irregular circus movements initiated in the auricular muscle and perpetuated by the occurrence of local blocks[1-6] but this view has been challenged by many authors[4, 2, 94] who believe that both auricular fibrillation and flutter are the result of stimuli arising in a single exciting ectopic focus According to the latter opinion all auricular arrhythmias have a similar mechanism the only difference between them being the number and frequency of impulses arising from the single focus[21-]

The paroxysmal form of auricular fibrillation usually sets in abruptly with a ventricular rate between 120 and 160 and a totally irregular rhythm A pulse deficit may be present When heart failure is absent it is best to observe these patients for several hours at rest and with sedation If the fibrillation persists 3 grains (2 gm) of quinidine should be given orally every two hours The first dose may be used to test sensitivity for reactions will usually occur within two hours if the patient is allergic to the drug[59] True sensitivity is unusual however and treatment of a patient who is critically ill should not be delayed If there is no change in rhythm after four hours the dose is increased to 6 grains (4 gm) every two hours It must be remembered that quinidine has relatively little cumulative effect and that peak plasma levels are reached after 4 or 5 adequate doses have been given[9] If the same dose is administered thereafter the plasma level rises only slightly and it is therefore necessary to increase the dose again to achieve

an effect The total daily or weekly dose is of much less importance in determining plasma concentration than is the amount of the individual dose[2,5] as only traces of quinidine are found in the plasma twenty four hours after the last dose has been given Occasionally it is necessary to raise the dose to 9 grains (6 gm) every two hours If regular rhythm is not restored with these doses larger ones will rarely have the desired effect and if the arrhythmia persists after 24 to 36 hours the drug should be discontinued

We have used quinidine intramuscularly during the past year with excellent results A suitable preparation is a 20 per cent solution of quinidine sulfate in propylene glycol 3 grains (2 gm) per cc The preparation is stable and the injection is rarely painful[94] The dosage is the same as when given orally and there is no greater toxicity when the drug is given by this route Intramuscular medication is especially valuable when oral administration cannot be tolerated Quinidine hydrochloride is also satisfactory for intramuscular use Intravenous quinidine which is now available as quinidine lactate in 10 cc ampules containing 65 gm is a dangerous drug and its use must be carefully controlled There is little indication for the intravenous administration of quinidine as most cases will respond to intramuscular or oral medication

If the fibrillation persists after a satisfactory trial with quinidine the patient should be digitalized (*vide infra*) In all recalcitrant cases it is essential to exclude the presence of hyperthyroidism or active rheumatic heart disease In

quinidine therapy after the hyperthyroid state has been corrected[4]

If quinidine restores sinus rhythm the dose is reduced gradually and a maintenance dose is instituted Three to six grains (2 to 4 gm) 3 times a day may suffice but 6 to 9 grains (4 to 6 gm) 4 or more times a day may be re

quired for long periods When quinidine is given q i d the first dose is taken preferably early in the morning on awakening the second at midday the third in the evening and the fourth dose just before retiring in order to maintain adequate blood levels In unusual instances the fourth dose must be given during the night

An example of this type of case is that of a fifty nine year old white male who had severe rheumatic heart disease and auricular fibrillation and was treated with 3 gm (5 gr) of quinidine sulfate orally every two hours as outlined above Regular rhythm was restored and he was maintained successfully on 3 gm 4 times a day awaking at 4 A M daily for the morning medication Whenever this dose was omitted fibrillation recurred

Recently 3 grains (2 gm) enseals of quinidine have been introduced to avoid giving a dose during the night The drug in this form is more slowly absorbed allowing for a greater interval between doses

If only one attack has occurred or when episodes are infrequent quinidine need be given only for one week After the acute attack however if paroxysms occur frequently prophylactic quinidine should be continued indefinitely 1

Occasionally the attacks respond to quinidine with increasing difficulty as they become more frequent In such cases moderate doses of digitalis given in addition to the quinidine are sometimes beneficial 1 When the attacks recur with increasing frequency and do not readily respond to quinidine it may be more advisable to slow the ventricular rate with digitalis than to attempt to restore sinus rhythm with quinidine A good example of this therapy is presented by a woman of forty four who had had rheumatic heart disease with mitral stenosis for twenty years She had developed a cerebral embolus three years before Since then she had experienced frequent auricular premature beats and episodes of auricular fibrillation At first these readily responded to quinidine but the attacks

became very frequent and lasted several days in spite of large doses of this drug The patient was much distressed during the attacks and developed great anxiety regarding their recurrence In the next attack digitalis alone was given until the ventricular rate fell to 70 Since then she has been maintained on 5 mg digoxin daily has remained in chronic auricular fibrillation but has been entirely comfortable

Although recent studies have shown that adequate cardiac output can be maintained by many patients with chronic auricular fibrillation if they are adequately digitalized [140] some of them do not do so well during fibrillation as when their rhythm is regular [9] Not only is their activity restricted but there is the ever present danger that thrombi will form in the auricles of the fibrillating heart and that embolization will occur For these reasons we believe that all attempts to restore sinus rhythm should be made before resorting to digitalis alone although this therapy may occasionally be satisfactory as illustrated in the above case

When employed properly quinidine is a safe and effective drug The harmful effects following its use have in general been overemphasized Several premonitory signs and symptoms of overdosage in patients under quinidine treatment should be kept in mind The most common of these may be grouped under the term cinchonism They include tinnitus impaired hearing headache blurring of vision giddiness nausea vomiting abdominal cramps and diarrhea If the drug is given too rapidly by the intravenous route respiratory depression and convulsions similar to those seen in animals may result [96] Rarely a true *idiosyncrasy may be present In such cases acute respiratory* distress and circulatory collapse appear even after small doses Serious skin eruptions[201] occasionally are a manifestation of quinidine sensitivity and rarely thrombocytopenic purpura develops (*vide infra*) [200, 23]

A dramatic instance of quinidine sensitivity was seen in I K a teacher of sixty-eight, who had had a coronary

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symptoms listed above become manifest, the drug should be discontinued If the patient is desperately ill, however, because of an arrhythmia, it may be necessary to continue quinidine administration despite mild symptoms of toxicity Even in these cases however, if severe symptoms of toxicity occur the drug should be discontinued and other medication given

Some authors have stated that quinidine should not be used in the presence of complete atrio ventricular or bundle branch block[51] but we have not hesitated to employ this drug in supraventricular arrhythmias with bundle branch block, and have not observed any serious reactions as a consequence of this therapy

When quinidine is unobtainable oral potassium (5 to 10 gm daily given every four hours) may convert auricular fibrillation to sinus rhythm[179], This therapy was employed during World War II with excellent results

In the treatment of acute paroxysmal fibrillation, occasional good results with Pronestyl[187] (*vide infra*) have been reported These results as yet, have not been confirmed This drug, however is not successful in converting a chronic fibrillation to regular sinus rhythm, although slowing of the ventricular rate does occur

If heart failure is present during the paroxysm of auricular fibrillation, digitalis should be administered immediately It increases the degree of A-V block and slows the ventricular rate, but does not affect the fibrillation The measures suggested below for the treatment of heart failure should also be instituted Digitalization can be accomplished in many ways with various preparations It is advisable to learn the potentialities of one preparation for oral use and of another for intravenous administration

If the patient is acutely ill and immediate digitalization is indicated, strophanthin (K) or Ouabain (G strophanthin) should be given intravenously These drugs have a very brief latent period, and are usually effective in from five to fifteen minutes, with a maximum effect in thirty to one

occlusion thirteen years ago and who recently experienced ventricular premature beats On several occasions he developed acute laryngeal edema after receiving 3 grains (2 gm) of quinidine twice a day A single daily dose finally sufficed to prevent the premature beats and no further episode of edema occurred while the patient was on this dose Another graphic demonstration of quinidine idiosyncrasy was that of S G a physician of fifty six with essential hypertension and coronary artery disease On several occasions during the years he had received several grams of quinidine for ventricular premature beats Six months ago he had taken 3 grains (2 gm) of quinidine 6 times during a two day period when he developed profuse ecchymoses in the skin and mucous membranes and profuse hematuria A blood count showed that the platelets had almost disappeared from the blood The quinidine was discontinued and recovery occurred spontaneously within three weeks These sensitivity reactions should be treated by immediate withdrawal of the drug Blood transfusions should be given if required and antihistaminic preparations may be of great value

Quinidine produces significant electrocardiographic changes widening of the QT interval being the most common finding This is due to widening of the RT segment and only occasionally to widening of the QRS complex[38] The latter indicates a toxic effect of quinidine These changes were noted in a thirty four year old man who was admitted to the hospital because of recurrent episodes of palpitation and was discovered to have a nodal tachycardia He was put on quinidine 6 grains (4 gm) every two hours After 8 doses an electrocardiogram showed a QT interval of 58 seconds largely as a result of widening of the RT segment When the quinidine was stopped the QT interval gradually returned to within normal limits within twenty four hours The QT interval may be even more prolonged than in the case cited above[2] If significant widening of the QT interval is found or if some of the

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hundred twenty minutes [66, 11] A safe initial dose is 0 3 mg , within half an hour 0 1 mg may be given and repeated every half hour or hour until the ventricular rate is slowed to 70 or less [76] Strophanthin K is available in 1 cc ampules containing 0 65 mg and Ouabain in 0 5 cc ampules containing 0 1 mg and 2 cc ampules containing 0 5 mg We have used both of these drugs intravenously with good results

Lanatoside C (Cedilanid) may also be given intravenously It has a slightly longer latent period and causes a decrease in heart rate in from twenty minutes to two hours [67] Its effect lasts longer than that of Strophanthin K or G making this drug ideal if moderately quick and slightly prolonged action is required The initial dose given is approximately one half the digitalizing dose *i e* 8 mg (4 cc) which is followed by 4 mg (2 cc) every four to six hours as required Cedilanid is available in ampules of *2 cc containing 4 mg and in ampules of 4 cc containing* 8 mg

It is important to remember that these preparations are eliminated fairly rapidly and that the digitalization effect will be lost if longer acting digitalis preparations are not administered as soon as practicable *i e*, within twenty four hours In this way digitalization is maintained Another very satisfactory method to follow if the patient is able to take medication orally is to give an initial intravenous dose and at the same time begin oral digitalization The intravenous medication is not repeated and oral digitalis is continued thereby giving rapid effect and prolonged action In general it is rarely necessary to use intravenous preparations except in patients with severe heart failure and rapid fibrillation

For oral use we have found Digoxin a glycoside of *Digitalis lanata* very satisfactory It has the advantage of being a pure substance of definite and constant composition and unlike digitalis leaf it is almost completely absorbed from the gastro intestinal tract [37] It is eliminated rapidly and is not as apt to cause cumulative toxic effects such as those

caused by more slowly excreted glycosides, e g , Digitoxin The effect of a single dose of Digoxin usually persists only for several days,[14] whereas the effect of Digitoxin may continue for two to three weeks[55] We believe that at the present time, Digoxin is a very safe and effective preparation for oral digitalization

The amount of any digitalis preparation required for digitalization varies greatly from person to person and the dosage must be individualized The digitalizing dose of Digoxin varies between 1 5 and 5 0 mg[76] An initial dose of 1 0 to 1 5 mg is given, thereafter, 0 5 mg is given every six hours until the desired effect is obtained The maintenance dose is 0 25 to 0 75 mg (1 to 3 tablets) daily, most patients being well controlled on 5 mg Frequently giving 25 mg and 50 mg on alternate days proves very satisfactory

Many physicians still use the whole digitalis leaf preparation for both digitalization and maintenance This preparation is a good one, but the delayed onset of its action and the long period required for its dissipation are usually disadvantageous When the patient will not take medication regularly, however, the use of the leaf is preferable, since the effect of Digoxin disappears in forty-eight hours and the patient may go into cardiac failure if he neglects to take it for two or three days This danger is less when digitalis leaf is used The whole leaf is much safer than Digitoxin and usually gives good results Initial digitalization with the leaf can be accomplished with a total dose of 1 to 1 5 gm (15 to 22½ grains) given over a period of twenty four to forty-eight hours, as desired A useful guide for dosage is to give 1 gm (gr 1½) per 10 pounds of body weight[60] Adequate maintenance is usually achieved with 1 to 3 grains (06 to 2 gm) daily

The patient should be observed carefully for signs of digitalis toxicity Not infrequently, the most important and earliest of these are abdominal distention, cramps or pain, and visual disturbances, such as yellow or green vision,

and scotomata.[71] Occasionally cerebral symptoms such as confusion, dizziness, and delirium appear. Nausea, vomiting, and diarrhea occur in most cases of digitalis overdosage but may be late manifestations.[19] The most significant sign of overdigitalization is an arrhythmia resulting commonly from ventricular premature beats which occur in pairs or triplets and produce bigeminal or trigeminal rhythm. Bradycardia and various degrees of A-V block may also occur. It must be emphasized that premature beats may be found in patients who are not nauseated and do not vomit, and if they occur the drug should be discontinued or the dosage decreased.

Depression of the RS-T segment and/or inversion of the T wave in the electrocardiogram indicates that digitalis has had an effect on the heart muscle but does not imply toxicity. In patients with hyperthyroidism, digitalis is usually ineffective even when tremendous doses are given. Conversely, when a patient with auricular fibrillation or congestive failure fails to respond to adequate doses of digitalis one should suspect the presence of hyperthyroidism. On the other hand, some people with hyperthyroidism have an increased susceptibility to digitalis and readily develop toxic symptoms.[86] Careful studies with radioactive iodine may reveal hyperthyroidism as the cause of certain instances of such digitalis "sensitivity."

The various arrhythmias resulting from digitalis overdosage usually disappear spontaneously; if Digoxin was used, they disappear within two to three days after the drug has been discontinued, but they may continue for two to three weeks if Digitoxin had been given. Usually no treatment is necessary for the arrhythmia except withdrawal of the drug.[7] In some instances of supraventricular tachycardias,[41] ventricular premature beats, or ventricular tachycardias[169] induced by digitalis, specific treatment may become necessary. *In these cases, potassium chloride or* acetate given orally usually eliminates the arrhythmias within *thirty minutes*[77,89] (Fig. 1). *Two to ten grams of* a









Fig. 1–58. Hypertensive and vascular disease. Digitalis toxicity. A, Lead III. 40 ventricular premature beats per 100 beats. B, Lead II, 15 minutes after 10 Gm. Potassium orally, 6 premature beats per 100 beats. C, Lead II, 30 minutes, 8 premature beats per 100 beats. D, Lead II, 45 minutes. No premature beats. (Courtesy of Dr. Charles Enselberg.)

20 per cent solution may be given in syrup of citric acid or smaller quantities may be given in milk, orange juice or ginger ale. When potassium is given in this manner nausea and vomiting usually do not occur. The effect produced will only last for from two to four hours and the treatment may have to be repeated. Toxic effects of potassium do not occur if there is adequate urinary excretion. Rarely is it necessary to use potassium intravenously; if it is, extreme caution should be used and only 0.5 to 1.5 gm should be given slowly. Electrocardiograms should be taken frequently to detect potassium toxicity. Marked peaking of the T waves, widening of the QRS complexes and disappearance of P waves are the most commonly observed changes when potassium intoxication occurs.[15]

Atropin is often effective in abolishing the A-V block caused by digitalis.

In auricular fibrillation secondary to hyperthyroidism or active rheumatic fever, digitalis is ineffective.[7] After the hyperthyroidism has been treated with radioactive iodine or by other methods, the arrhythmia often disappears spontaneously[93] or responds to quinidine therapy.[74] A case recently studied illustrates this point. A fifty-three-year-old female complained of constant palpitation. She had been found to have auricular fibrillation and had received very large doses of digitalis and quinidine without abolition of the arrhythmia or slowing of the ventricular rate. Although clinical signs of Graves' disease were absent, only 20 per cent of a test dose of radioactive iodine (I_{131}) was excreted and a diagnosis of hyperthyroidism was made. A therapeutic dose of 5 millicuries of I_{131} was administered. Two days later she was started on 30 drops of Lugol's solution daily. This medication was continued for four weeks and, while the fibrillation persisted, the ventricular rate fell to 84. The arrhythmia was still present six weeks after the administration of I_{131} and for this reason 6 grains of oral quinidine were given every three hours. Sinus rhythm ap

peared after 8 doses and persisted even after quinidine was discontinued

Radioactive iodine is usually picked up by the thyroid gland within twenty four hours but its effect on metabolic processes may not become evident for four to six weeks [26] It may be necessary therefore as in the case cited to administer Lugol s solution (10 drops t i d) during this period This medication may be started twenty four hours after radioactive iodine has been given and should be continued for approximately three weeks [7] The effect of the Lugol s solution will last for another week at which time the radioactive I_{131} effect will have begun to manifest itself

If propyl thiouracil alone is used in hyperthyroid patients an effect on the arrhythmia should be seen within two to four weeks [7] It should be emphasized that the prognosis in these cases of auricular fibrillation due to hyperthyroidism is excellent if the primary disease is controlled [17]

Cases of auricular fibrillation occurring during active rheumatic fever usually respond to digitalis or quinidine therapy when the active process has been controlled Failure to respond to digitalis suggests that the underlying disease is still active The prognosis in these cases is not nearly as good as in patients with hyperthyroidism especially if a mitral valvular lesion is present [20]

AURICULAR FLUTTER

In auricular flutter, the ventricular rhythm is usually regular with a rate of 125 to 180 but it may be irregular because of varying degrees of A V block and thus may resemble auricular fibrillation Auricular flutter may sometimes be distinguished from fibrillation by the observation of rapid regular venous pulsations in the neck at a rate of over 250 corresponding to the rate of the flutter waves in the electrocardiogram Usually the atrial rate varies between 250 and 350 and a 2 1 A V block is present When auricular flutter is complicated by a bundle branch block

pattern it may simulate ventricular tachycardia electrocardiographically (Fig 2) Occasionally auricular flutter can be diagnosed with certainty only in the electrocardiographic leads taken to the right of the sternum [5] or from the right chest or back In rare instances it is necessary to take esophageal leads to record the auricular waves [42]

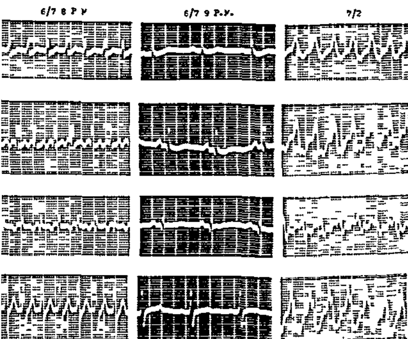


FIG 2 — S W m 71 Auricular flutter treated with intramuscular quinidine 0 6 Gm and oral Digoxin 0 8 mg 6/7 8 P M 2 1 auricular flutter 6/7 9 P M 1 hour after therapy regular sinus rhythm 7/2 2 1 auricular flutter with bundle branch block simulating ventricular tachycardia

Auricular flutter is usually paroxysmal but it may become chronic and continue for months or years [145] If a paroxysm does not remit spontaneously after several hours an attempt should be made to stop the attack with digitalis

or quinidine (Fig 2) digitalis being employed in all cases with heart failure Digitalis slows the ventricular rate and increases the efficiency of the heart

The fact that digitalis will oftentimes convert auricular flutter to fibrillation and that restoration of sinus rhythm will occur when the drug is discontinued has been repeatedly stressed[16] since the original studies in 1911[157] Although recent observations[25] have cast some doubt upon this theory we have seen a great many patients follow this pattern when digitalis is administered //It has been our policy to administer digitalis convert flutter to fibrillation and then stop the digitalis in the hope that sinus rhythm will result If sinus rhythm does not occur quinidine is employed to restore the normal rhythm Usually a small dose of quinidine will be effective Other observers also favor this approach to the therapy of auricular flutter[250 266] Digitalis and quinidine are employed as described under auricular fibrillation In critically ill patients there should be no hesitation about using intravenous digitalis followed by intramuscular quinidine if necessary

In some instances auricular flutter persists in spite of all these measures and the patient must be maintained on digitalis as in chronic auricular fibrillation in order to keep the ventricular rate between 65 and 75 beats per minute Some patients may continue to have auricular flutter for years and then suddenly remit spontaneously[235]

If attacks of paroxysmal auricular flutter recur frequently an attempt should be made to prevent them by the continuous use of quinidine The dose of quinidine for maintenance is 3 to 6 grains (0 2 to 0 4 gm) 4 times a day In some cases however we have found it necessary to increase the dose and the frequency of administration giving as much as 9 grains (6 gm) 4 or 5 times a day If this is not effective digitalization should be carried out and a maintenance dose continued In this way a certain degree of A V block is maintained and when an attack occurs the ventricular rate may not markedly increase despite a rapid

auricular rate This may serve to keep the patient symptom free during an attack. This method is not successful in all cases for, despite adequate digitalization, a tachycardia may develop and symptoms appear

AURICULAR AND NODAL TACHYCARDIA

Auricular and nodal tachycardia cannot be distinguished clinically and are treated alike These tachycardias are frequently functional and may be precipitated by tobacco alcohol infections, gastric distention Graves disease, allergic reactions and tense emotional states Very frequently there is a combination of causes The attack generally begins suddenly although in certain instances there may be a short premonitory period The ventricular rate usually is regular, despite changes of position or exercise,[79] and is between 180 and 220 beats per minute, which is somewhat more rapid than in auricular flutter Although there is usually a 1 1 ventricular response, occasionally a 3 1 or 2 1 response occurs with a resultant ventricular rate between 75 and 100[56] These cases are more common than was previously suspected (Fig 3) Frequently a supraventricular tachycardia can be differentiated from auricular flutter by the absence of flutter waves in the cervical veins Unlike cases of auricular flutter, in which only transient slowing of the ventricular rate may be noted following vagal stimulation, supraventricular tachycardia often is terminated by this procedure Supraventricular tachycardias are particularly apt to be evanescent, and patients may have many attacks daily for years without experiencing difficulty Patients subject to recurrent attacks have learned, through experience, that certain maneuvers stimulating the vagus nerve abolish an attack, *e g*, sudden movements of the head holding the breath, coughing vomiting eating lowering the head over the bed, or bending forward in a chair

Often the attack disappears spontaneously but, if it persists, an attempt should be made to terminate it by



Fig. 3 — P E m 52 Coronnry nrterv disease Paroxysmal auricular tachycardia 190 ventricular rate 95

mediately when given by vein Mecholyl should not be given to allergic or asthmatic patients because of the danger of inducing bronchial spasm or to hyperthyroid patients because auricular fibrillation may be precipitated [194] It is best to keep the patient in a horizontal position when this drug is used to avoid possible syncope Mecholyl and carotid sinus pressure usually are ineffective in cases of auricular tachycardia with A V block and in auricular flutter and fibrillation and they may be ineffective if quinidine has been administered previously [40]

A less toxic parasympathomimetic drug which is effective in the treatment of supraventricular tachycardias is *acetylcholine* [231] The effect of this drug is brief and its side effects are less marked than those of Mecholyl Although we have had little experience with this preparation other observers have found it to be satisfactory when given in a single intravenous dose up to 100 mg Certainly acetylcholine deserves a more extensive clinical trial

Neostigmine another parasympathomimetic drug is also effective in abolishing supraventricular tachycardias and is given in doses of 1 mg intramuscularly [36] When any one of these drugs is used atropine should be available for immediate use should it be necessary

In cases in which mechanical methods are unsuccessful in which the distressing side effects of Mecholyl preclude its further use and in which more rapid action than is usually provided by quinidine is desired *neosynephrine* should be employed When given intravenously in doses of 5 to 1 0 mg., this drug usually stops an attack of supraventricular tachycardia within twenty to thirty seconds [275] It produces a rise in blood pressure stimulation of the cardio inhibitor fibers in the aortic arch and carotid body and reflex cardiac slowing Most attacks revert when the systolic blood pressure has risen to 160 mm and the pressure usually returns to normal within ten minutes or less There are few toxic effects of the drug but it should not be used in patients whose blood pressure is elevated during an attack or in

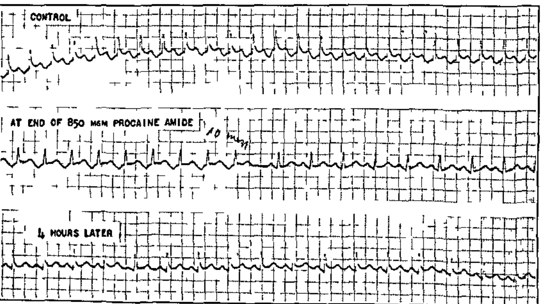


Fig 4 — M B m 45 Paroxysmal nodal tachycardia of unknown etiology Cessation 10 min following 0 850 gm procaine amide intravenously Continuous lead II (Kayden Steele Mark and Brodie Courtesy of Circulation)

cases with ventricular premature beats We have found neosynephrine effective in several cases after all other methods had failed

As in the case of paroxysmal auricular fibrillation and flutter of recent origin Pronestyl occasionally stops auricular or nodal tachycardia [154] Figure 4 shows the rapid effect of 0 8 gm of the drug given intravenously to a man with nodal tachycardia This patient of forty five developed repeated episodes of this arrhythmia but no cardiac disease could be demonstrated

It should be remembered that supraventricular tachycardias are so frequent and so often remit spontaneously that the accurate appraisal of the efficacy of any drug in their treatment is difficult If there are numerous recurrences of the tachycardia the frequency of the attacks may be diminished by the continued use of quinidine or digitalis

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is infrequent and is usually associated with severe myocardial damage particularly that caused by coronary artery disease It may be seen in cases of rheumatic heart disease during cardiac catheterization and in normal individuals [5] Occasionally it is possible to differentiate ventricular tachycardia clinically from auricular tachycardia by a slight irregularity in rhythm and a variation in the intensity of the first heart sound Prominent jugular vein pulsations representing a summation of the auricular systolic wave (A wave) and the ventricular systolic wave (C wave) may be observed at a rate slower than that at the apex [91] although this is not a common finding

Digitalis is often a factor in the production of ventricular tachycardia [65] Some observers believe that all cases of the bidirectional type of this arrhythmia are secondary to digitalis overdosage [162] We have stressed the fact that digitalis *should be discontinued in all patients who develop ventricu*

lar premature beats especially if they are multifocal If this precaution is taken many cases of ventricular tachycardia can be prevented

Ventricular tachycardia usually responds readily to quinidine, given orally or intramuscularly occasionally an intravenous dose 1 gm (15 grains) has been required to stop an attack[21] We believe however that quinidine should be used intravenously only in dire emergencies because of its toxicity when given by this route and because better safer preparations are now available for the treatment of ventricular tachycardia

A procaine derivative *procaine amide, (Pronestyl)* has been introduced for the treatment of ventricular arrhythmias[167] This drug is a direct myocardial depressant It is supplied in 250 mg capsules for oral use and in ampules containing 100 mg per cc for intravenous administration We have used it with excellent results in ventricular tachycardia and premature beats including cases in which quinidine has failed Pronestyl is given orally in doses of 500 mg every three to four hours or intravenously in doses of 250 mg to 1 gm at a rate of 100 mg per minute every thirty to sixty minutes until an effect is obtained An electrocardiogram should be taken while Pronestyl is being given intravenously as the drug is often effective immediately If a change in rhythm or widening of the QRS complex occurs the drug should be stopped Occasionally oral doses up to 6 or 10 gm are required We have found that both the intravenous and oral routes are satisfactory for the treatment of the acute episodes but the intravenous administration of Pronestyl produces a much more rapid effect Ventricular tachycardia may be converted to regular sinus rhythm in from thirty seconds to two minutes following as small a dose as 250 mg of Pronestyl A case recently observed is of interest to illustrate the effect of Pronestyl

A sixty six year old white male experienced an attack of squeezing severe chest pain radiating to the left shoulder and arm, ten days prior to hospital admission Four days

prior to admission he developed dyspnea on the day of admission he became dizzy and weak noted palpitations and fainted On admission he was in acute pulmonary edema and was given 3 mg strophanthin K intravenously

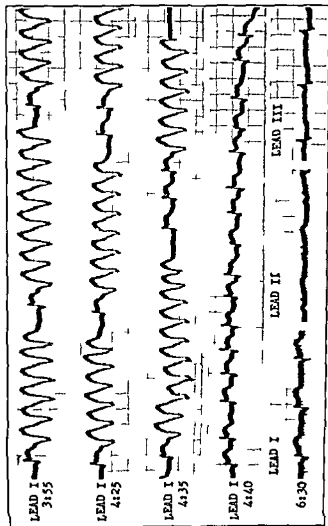


Fig 5 — M Y m 66 Acute Coronary Occlusion Precordial pain heart failure and occasional palpitation on 10 days Strophanthin K 0 4 mg just prior to admission Pronestyl 1 0 gm orally 3 45 P Digitoxin 1 0 mg orally at 6 P

morphine and 2 cc Mercuhydrin intramuscularly The electrocardiogram showed runs of ventricular tachycardia and acute anterior wall infarction (Fig 5) He was given 1 gram of Pronestyl by mouth and normal sinus rhythm developed within an hour (Fig 5) The Pronestyl was continued in doses of 1 gram every four hours for two days and then in doses of ½ gram every four hours for eight days He was also digitalized with Digoxin kept on maintenance doses and made an uneventful recovery

Although Pronestyl generally does not produce serious reactions it may cause a marked fall in blood pressure or even cardiac standstill when it is administered intravenously This may be prevented or counteracted by Neosynephrine (1 to 5 mg) intramuscularly The injection should be given slowly and blood pressure readings should be made during the injection When the drug is given orally in large doses it may produce nausea and other gastric symptoms making it necessary to stop the drug or reduce the dose We observed one patient who developed agranulocytosis while receiving Pronestyl and this has recently been pointed out by others Therefore frequent blood counts should be made and evidence of purpura or bleeding watched for

Pronestyl is also invaluable for the prevention and treatment of ventricular arrhythmias associated with operative procedures [134] and may be of value in intracardiac catheterization In preparation for the latter procedure it can be given prophylactically and if necessary during and after the manipulation If an arrhythmia occurs during cardiac catheterization despite this therapy the catheter should be partially withdrawn and should not be advanced until the arrhythmia disappears If used preoperatively 1 to 2 gms are given orally forty five to sixty minutes prior to surgery and 1 gm is given intravenously as the incision is made During anesthesia and operation intravenous procaine alone may be used in a dose of 5 cc of a 1 to 2 per cent solution to prevent ventricular arrhythmias [49]

Pronestyl has now been used prophylactically by us in a small series of patients who had previously experienced repeated bouts of ventricular tachycardia Although this drug is able to terminate the acute attacks quickly and effectively it does not appear to be completely effective in preventing recurrence of attacks when used orally 3 to 4 times daily It may be that the dose employed (500 mg t i d or q i d) is not adequate and patients with repeated attacks should perhaps be given much larger doses At the present time the best procedure appears to be to give Pronestyl for the acute episode and quinidine prophylactically

Many other drugs that have been used in the treatment of ventricular tachycardias deserve mention at this time One of these agents intravenous *magnesium sulfate* is not widely used but has proven effective in occasional cases [33, 40] When given rapidly in 2 to 4 gm doses *i e* 10 to 20 cc of a 20 per cent solution this drug may terminate an attack after other measures have failed Transient unpleasant side effects such as nausea flushing weakness and dizziness may appear It should be reserved for use after other therapy has proven ineffective With the newer agents available little opportunity for the use of $MgSO_4$ will present itself Atabrine in doses of 4 gram intramuscularly may occasionally be effective [93] Intravenous morphine sulfate 10 to 20 mg (1/6 to 1/3 gr) repeated every hour if necessary has been used successfully by several different observers [99, 11] The use of potassium salts (citrate or acetate in syrup or water or chloride as enteric coated tablets) orally either alone in doses of 1 to 5 gms 2 to 4 times daily or in conjunction with quinidine has recently been advocated for the treatment of refractory cases or to diminish the frequency of recurrences of ventricular tachycardia [245] We have found potassium to be particularly useful in the treatment of ventricular tachycardia secondary to digitalis overdosage and in patients who are sensitive to quinidine

Dibenamine hydrochloride an effective adrenergic blocking agent has not as yet been used by us in the treatment of tachycardias This drug has been shown to be most effective in blocking arrythmias which occur during cyclopropane anesthesia [131 175 198] and deserves a clinical trial in ventricular tachycardia which is not precipitated by operative procedures

VENTRICULAR FIBRILLATION

Ventricular fibrillation is rarely diagnosed clinically and *although it probably accounts for some cases of sudden* death it is difficult to state its exact incidence It occurs particularly in association with myocardial infarction anesthesia, or excessive amounts of digitalis The patient becomes pulseless and unconscious and rapidly goes into shock Convulsions occur if this arrythmia persists for more than thirty seconds The electrocardiogram shows bizarre ventricular complexes and a completely irregular rhythm Clinically, these attacks resemble a Stokes Adams seizure secondary to complete heart block and cardiac standstill *and it is important to distinguish between these two conditions electrocardiographically* The use of sympathomimetic drugs *i e* epinephrine or ephedrine so valuable in the treatment of cardiac asystole is contraindicated in ventricular fibrillation If possible the physician should obtain an electrocardiogram during the attack to establish its exact mechanism Once an episode is diagnosed correctly as ventricular fibrillation it can be assumed that subsequent attacks will be caused by a similar mechanism and not by cardiac standstill and they may be treated accordingly Bouts of both ventricular fibrillation and cardiac asystole may however occur in the same patient (Fig 6)

Since an immediate effect upon the arrythmia is necessary intravenous quinidine should be given promptly as quinidine lactate 4 to 6 gm (6 to 9 gr) despite the fact that the use of this route of administration is not without danger Magnesium sulfate, 10 cc of a 20 per cent solution, may also be effec

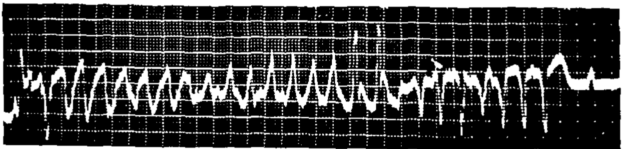


FIG 6—M N m 60 Coronary artery disease Complete A V block with periods of ventricular fibrillation and Stokes-Adams seizures Lead II continuous

TABLE 1.—DIFFERENTIAL DIAGNOSIS AND TREATMENT OF PAROXYSMAL TACHYCARDIA*

Type of Arrhythmia	Onset	Other Diseases Present	Signs—Heart Rate Pulse Rate Symptoms	Electrocardiogram	Treatment	Prognosis
A) Acute Paroxysmal Auricular Fibrillation	Sudden, may occur postoperatively or following infections	Rheumatic heart disease, hypertensive cardiovascular disease, arteriosclerotic heart disease, Graves' disease, and occasionally normal	Heart rate 120–190, pulse deficit may be present. Pulse rate 70–1[illegible]0 or more, completely irregular	1) No P waves, completely irregular rhythm	Digitalis, Quinidine, Pronestyl (see text)	Depending upon primary condition; normals—good. Rheumatic heart disease may be fair to poor. Arteriosclerotic heart disease—depending upon age, presence of congestive failure. Graves' disease excellent after primary condition is corrected
B) Auricular Flutter	Sudden	Most common is organic heart disease, rheumatic heart disease and arteriosclerotic heart disease	Pulse rate 125–1[illegible]0 depending on degree of heart block; rate may vary from minute to minute if degree of block changes. Flutter waves in neck at rate of 250–3[illegible]0	2) Flutter waves at rate of 250–350. Usually regular rhythm	Digitalis until fibrillation, then stop or continue to keep slow rate, or Quinidine (see text)	Same as fibrillation
C) Supraventricular Tachycardia 1) Nodal Tachycardia 2) Auricular Tachycardia	Usually sudden and usually no premonitory symptoms. May occur postoperatively or following infection	Patients usually normal. Nervous individuals	Sensation of palpitation, dizziness and precordial pain may occur. Rate 140–2[illegible]	3) Absolutely regular rhythm. P wave present. Short P-R interval. P waves may occur after QRS complex	Vagal stimulation by carotid sinus or eyeball pressure. Drugs: Mecholyl, Acetylcholine if no effect, Quinidine. Neosynephrine may be effective	Frequent recurrence can be controlled by prophylactic Quinidine
D) Ventricular Tachycardia	Sudden	Usually patients with organic heart disease; occasionally occurs on Digitalis intoxication	Fainting not uncommon. Pulse rate 1[illegible]0–2[illegible]0. Blood pressure low to shock level. Variation of intensity of first heart sound. Rarely prominent neck pulsations	4) QRS abnormal and widened. Rate slightly irregular	1) Pronestyl [illegible] or by mouth 2) IM Quinidine 3) Potassium	Attacks can usually be stopped but eventual prognosis is poor
E) Ventricular Fibrillation	Sudden	Usually patients with organic heart disease, arteriosclerotic heart disease	Stokes-Adams seizures common. Pulse unobtainable. Heart rate not obtainable. Blood pressure shock levels	5) Extremely bizarre complexes	[illegible] Quinidine, [illegible] MgSO4, [illegible] Atropine, [illegible] Pronestyl	Extremely poor. Often pre-terminal

* See [illegible]

tive Other drugs have been suggested for the treatment of this condition but have had limited trial Intravenous dibenamine has been shown to be effective in preventing ventricular fibrillation in animals and may prove to be of value in human beings Intravenous atropine, 2 mg (gr 1 30) has been used successfully to abort the acute attack and smaller doses 5 mg (gr 1 100) intramuscularly have been given daily to prevent recurrent bouts in patients subject to paroxysmal ventricular fibrillation [207] Intravenous Pronestyl has not been used by us for this condition but it should prove to be of value In many cases the attack will cease or the patient expire before therapy can be instituted

HEART BLOCK

Complete heart block with sudden failure of the idioventricular pacemaker and asystole is the commonest cause of Stokes Adams syndrome although a similar clinical picture may be seen in cases of ventricular tachycardia or other arrhythmias [203] Heart block is usually seen in patients with organic heart disease secondary to coronary arteriosclerosis but a small percentage of cases results from digitalis overdosage [12] Congenital heart block is much more common than was previously believed and may account for 10 per cent of all cases [69, 5] Many patients experience only momentary weakness or unconsciousness and require no therapy but others have severe symptoms and require immediate treatment One half to one cc of a 1 1000 solution of epinephrine is given subcutaneously or directly into the heart in extreme cases in shock If the seizures recur the Adrenalin should be repeated subcutaneously every hour or two or may be given in oil (1 cc = 2 mg) intramuscularly every twelve to twenty four hours We have observed a fifty six year-old female who required 5 cc of Adrenalin every fifteen to thirty minutes for hours in order to remain free of attacks Intracardiac Adrenalin was necessary on one occasion after intramuscular therapy

had failed and during a period when the patient experienced recurrent Stokes Adams seizures a slow continuous intra venous injection of 1 100 000 Adrenalin at a rate of 2 0 cc (30 drops) per minute was maintained for several hours in order to stop the syncopal episodes In patients who experience frequent episodes of Stokes Adams seizures ephedrine gr $\frac{1}{4}$ to $\frac{1}{2}$ (15 to 30 mg) or paredrine gr $\frac{1}{3}$ to 1 (20 to 60 mg) may be given orally 3 to 5 times daily in an effort to decrease the number of recurrences [97] In all cases where digitalis may be a contributing factor this drug should of course be withdrawn

OTHER ARRHYTHMIAS

Sinus bradycardia sinus tachycardia and *premature beats* may occasionally cause severe difficulty These will be discussed briefly

Sinus tachycardia is usually associated with febrile or toxic conditions congestive heart failure or emotional or metabolic disturbances There is no specific therapy other than that directed at the primary condition

Sinus bradycardia, like sinus tachycardia may be an incidental finding especially in athletes and pregnant women and usually requires no treatment It is caused by an increase in vagal tone or by a diminution in sympathetic tone or both [84] If the rate falls below 40 dizziness and syncope may result as in a person with carotid sinus syndrome Actual cardiac standstill may occur [207] We have recently observed a basketball coach of thirty two who usually had a pulse rate of 48 beats per minute He fainted on several occasions during tense moments in a game During the attacks of syncope his pulse rate fell to 30 and he usually recovered after thirty to forty seconds Usually these cases respond to ephedrine in 25 to 45 mg (3 8 or 3/4 gr) doses or to atropin sulphate (6 to 1 2 mg) gr 1/100 to gr 1/50 by mouth It may be necessary to give

atropin (6 mg to 2 mg) gr 1/100 to 1/30 subcutaneously or intravenously

Premature beats may cause palpitation precordial discomfort and anxiety particularly if they occur frequently These symptoms can usually be relieved by reassurance rest and sedation but occasionally they are sufficiently severe and persistent to require active treatment A definite relationship has been found between anxiety states and episodes of premature beats and it has been shown that they as well as the distressing symptoms which occur during attacks may in some cases be eliminated by adequate psychiatric therapy [272] If drugs are necessary to stop premature beats the physician should determine the site of origin of the ectopic beats before beginning therapy Quinidine is usually successful in reducing the frequency of both ventricular and auricular premature beats Pronestyl (procaine amide) is most effective in stopping or preventing ventricular premature beats

A forty two year old salesman began to experience frequent choking sensations associated with a momentary cessation of the heart beat and on several occasions he became faint and almost collapsed Examination showed the heart to be normal but the electrocardiogram revealed ventricular premature beats He received 250 mg of oral Pronestyl 4 times a day with prompt disappearance of the arrhythmia and has remained symptom free since that time

Although this patient was controlled by 250 mgm of Pronestyl q i d most commonly 500 mg is required orally every four to six hours to attain a similar effect Pronestyl is preferred to quinidine which was formerly employed and is more effective than is quinidine Premature beats assume great clinical importance when they precede the onset of paroxysmal tachycardia especially during operative procedures or following a coronary occlusion

Chapter

2

ACUTE PULMONARY EDEMA AND CONGESTIVE HEART FAILURE

MUCH uncertainty exists today concerning the patho logic physiology of heart failure and acute pulmonary edema since many previously accepted concepts of cardiovascular and renal physiology and of sodium metabolism have been discarded, and since new concepts have been introduced during the last ten years

Pathogenesis of Cardiac Failure —In the past the symp toms and signs of congestive failure were attributed to "backward" failure *i e*, myocardial insufficiency, (cardiac failure) with increased venous pressure and venous con gestion, increased blood volume secondary to anoxia of the bone marrow and resulting edema.[239] However, recent investigations have shown that this mechanism does not explain many of the findings in congestive failure Rather do they support the theory of ' forward failure, *i e*, cardiac insufficiency, diminished cardiac output with diminished renal blood flow, and decreased sodium excretion, with resulting increase in blood volume and edema [185] Most of the symptoms and signs found in congestive failure can be accounted for by these sequential events although they do not explain some of the findings in acute heart failure, *i e*, acute pulmonary edema (*vide infra*) Usually, when the cardiac output falls, there is a liberation of excessive amounts of antidiuretic hormone from the posterior pituitary gland[24] and of desoxycorticosterone from the adrenal cortex,[205] these, also, play a part in the retention of sodium and water, and contribute to edema formation Although the patho-

genesis of congestive heart failure has not yet been completely clarified it is probable that elements of both the forward and backward hypotheses play a part in its development

Excellent reviews dealing with the mechanisms of cardiac failure have been written [202 219 241] We wish however to emphasize certain facts presented in these papers concerning the patho physiology of the various types of heart failure A knowledge of these basic facts is essential for the intelligent handling of both the acute and chronic forms of this condition

Heart failure occurs when the myocardium is no longer able to pump sufficient blood to all the tissues to satisfy their metabolic requirements At first the diseased myocardium is able to respond to stresses by increasing the length of its muscle fibers and contracting more forcefully (*Starling s law of the heart*[239]) *Eventually however the* fibers are distended beyond physiological limits and are no longer able to maintain an adequate output for tissue needs

In congestive failure the cardiac output may be low or normal as in hypertensive arteriosclerotic or rheumatic heart disease or it may be normal or high as in thyrotoxicosis [242] anemia [242] beri beri [4] Paget s disease [83] arteriovenous aneurysm [45] or chronic pulmonary disease [219] In these latter conditions although the output is not decreased it still is unable to meet the tissue needs [243] A primary object in therapy therefore is to increase the cardiac output In cases of failure with low cardiac output this can often be done by decreasing the blood volume by means of salt restriction or phlebotomy The resultant diminished venous return and lowered venous pressure enable the overdistended inefficient cardiac muscle fibers to shorten to within physiological limits to contract with greater force and to increase stroke volume and output In cases of failure with high output correction of the primary condition usually brings relief Digitalis increases cardiac output in both types of heart failure although it is more effective in cases with low

output [41] In some cases of high output failure i e cor pulmonale, digitalis may even be harmful if given too rapidly It probably acts directly on the heart muscle increasing the force of its contraction [46 97] although a primary effect on the venous system has been suggested [60] This observation has not been confirmed

Sodium retention is probably the single most important factor in the production of the symptoms of congestive failure This retention of sodium and the consequent increase in total blood volume has been attributed to diminished cardiac output diminished renal blood flow[190] and decreased renal excretion of sodium and water [185] A patient with heart disease may remain well compensated while his sodium is restricted, but may quickly go into cardiac failure if given salt, because the resultant marked increase in blood volume throws an additional load upon the already overburdened heart [193] Adequate salt restriction with the use of mercurial diuretics to aid sodium excretion, is therefore, of prime importance in the management of congestive failure

A marked increase in body activity increases the work of the heart It may cause a patient who was well compensated during rest or mild activity, to go into severe cardiac failure It is important, therefore, to regulate a patient s activity carefully in order to avoid sudden episodes of cardiac failure

If the above principles are borne in mind in the course of treatment of congestive failure many lives will be saved and careless mistakes avoided, for example, phlebotomy is of great value in acute left heart failure secondary to hypertensive heart disease,[123] *i e* cardiac failure with a low output, but it may cause great harm if performed in other forms of heart failure *e g*, heart failure associated with chronic pulmonary disease, Graves' disease, or chronic anemia In these latter conditions a high output is necessary to maintain adequate tissue oxygenation and measures that lower the blood volume, such as phlebotomy, should

not be used since they further decrease the amount of oxygen available to the tissues

Decreased renal blood flow and sodium retention account almost entirely for the symptoms and signs of chronic congestive heart failure *i e* dyspnea, orthopnea, pulmonary congestion, distention of the neck veins, hepatomegaly, ascites, and peripheral edema Additional factors must be sought, however to explain the sudden occurrence of acute left heart failure, *i e* cardiac asthma and acute pulmonary edema

Paroxysmal Nocturnal Dyspnea —Many patients with mild left ventricular failure experience recurrent episodes of paroxysmal dyspnea several hours after they go to bed They awaken suddenly and are acutely dyspneic a few rales and pronounced wheezes over both chests appear, without other evidence of failure The blood pressure is high and the pulse is strong This paroxysmal nocturnal dyspnea commonly called *cardiac asthma*, is probably the result of a redistribution of blood and an increase in the blood volume with hemodilution when the patient assumes the recumbent position During the day blood is pooled in the splanchnic and peripheral areas, but when the patient lies down blood is redistributed to the thorax [64] which results in lung congestion As a result of this congestion, nerve reflexes from the lungs are initiated, and bronchospasm, dyspnea and the other symptoms and signs of cardiac asthma result The exact mechanism of this condition has not been completely clarified)

Complete relief is often obtained by having the patient sit up or stand, or walk about if possible Mild cases may be controlled by aminophyllin suppositories (5 gm or 7½ grains) Aminophyllin is safe when thus administered, and is completely absorbed Large doses can be given without producing significant gastric upsets In the more severe cases or in cases that do not respond to this simple measure, morphine sulfate should be given intramuscularly, 15 mg (1/4 gr), or intravenously, 8 to 10 mg (1/8 to 1/6 gr)

when an immediate effect is desired. The intramuscular route is usually satisfactory. Morphine very effectively suppresses the pulmonary reflexes that serve to perpetuate symptoms and should be used liberally.

An attempt to decrease venous return by the use of tourniquets, i.e., bloodless phlebotomy, should be made if morphine is ineffective. Peripheral resistance, which is high in many of these cases because of sympathetic nerve discharge, may be decreased by giving aminophyllin (0.5 gm intravenously).[19] Digitalization may not be necessary in the milder forms of cardiac asthma. Recurrences can be prevented by means of a low sodium diet and mercurial diuretics and by having the patient sleep on three or four pillows or in a chair in the severe cases. If attacks recur in spite of these measures or if other signs of left and or right heart failure occur, the patient should of course be digitalized.

Acute Pulmonary Edema. The onset of acute pulmonary edema secondary to left ventricular failure may be extremely sudden with signs of circulatory collapse, severe dyspnea and cyanosis. It may occur following exercise, a large meal, paroxysmal tachycardia, a coronary occlusion, trauma to the central nervous system, or after severe emotional upsets. A discharge of impulses from the sympathetic nervous system has been suggested as a possible explanation in the latter conditions.[161]

A good example of pulmonary edema induced by neurogenic factors was recently provided by a 65 year old woman who had experienced occasional attacks of pulmonary edema three years before but had been quite well since then. One week ago she witnessed her husband fall and severely injure his scalp as a result of a cerebral hemorrhage. He quickly developed a left hemiplegia. His wife appeared to be taking all this well but about an hour later she suddenly began to wheeze and numerous rales appeared in both lungs. She was given Demerol 50 mg subcutaneously and gradually improved.

Occasionally pulmonary edema may complicate bulbar

poliomyelitis and for this reason intravenous hydration should be carefully supervised in these cases[118, 761] The heart sounds may be obscured by numerous bubbling rales in the lungs usually numerous wheezes are also present In many cases the right heart is strained by the failing left ventricle and the pulmonic second sound is accentuated If the patient is seen early the blood pressure and pulse volume may be maintained A short time after the onset of symptoms however there is a marked fall in blood pressure and the pulse becomes rapid and thready *i e* shock occurs due to acute left ventricular failure *Morphine sulfate* should be given intramuscularly at once or intravenously in cases with peripheral collapse in which little or no absorption would otherwise occur The doses employed are similar to those given in cardiac asthma This drug usually produces a dramatic improvement but if no effect is noted it should be repeated in fifteen to twenty minutes Dilaudid 1.5 to 3 mg (1 40 to 1 20 grain) or Pantopon 20 to 30 mg (1 3 to 1 2 grain) may be used instead of morphine especially if the latter induces vomiting Although some physicians use atropine 0 4 to 0 6 mg (1/50 to 1/100 gr) routinely in the treatment of acute pulmonary edema we do not feel that it is of value in cases in which tachycardia is present it may actually be harmful

Despite the fact that the arterial blood is fully oxygenated in many cases *Oxygen* is of value in pulmonary edema[9] Often when given by the meter mask under pressure (3 to 6 cm) it produces dramatic results A thirty year old negress para III with rheumatic heart disease experienced mild toxemia of pregnancy She was delivered of a full term normal infant after a short labor but bled profusely and rapidly went into shock She received 500 cc of plasma and 2500 cc of blood within the next three hours after having lost an estimated 2000 cc of blood She recovered from shock but developed a marked pulmonary edema one hour later Despite repeated doses of morphine oxygen by tent 8 mg (4 cc) Lanatocide C and continuous bloodless phlebotomy

with tourniquets (*vide infra*) she did not improve After one hour of vigorous therapy oxygen under pressure (4 cm) was administered by the anesthetist and within 10 to fifteen minutes marked improvement occurred The rales gradually disappeared and the patient recovered

To prevent recurrences oxygen may be given in this way for a period of fifteen minutes every few hours for the following twelve hours Oxygen by nasal catheter usually is not satisfactory but an oxygen tent often is of value in warm weather It should be remembered that the percentage of oxygen inspired by a patient when a nasal catheter or tent is used is never as great as that inspired through a mask (35 to 65 per cent as compared to 50 to 100 per cent) There is some evidence that 100 per cent oxygen may be irritating to the lung parenchyma particularly if taken for hours or days for this reason the use of a lower concentration is advisable [6]

Intravenous aminophyllin 0 5 gm (7½ gr) is of particular value in the treatment of acute pulmonary edema when Cheyne Stokes respiration is present When given by vein it should be administered very slowly the total dosage being injected in forty five to sixty seconds In less serious cases the same amount of aminophyllin may be given by suppository

If the pulmonary edema persists after the above measures have been tried 25 mg of Strophanthin K or Ouabain (Strophanthin G) and 2 cc of a mercurial diuretic Mercuhydrin or Mercupurin should be given intravenously in the same syringe Thereafter the Strophanthin is repeated or Digoxin is used as outlined previously Mercurial diuretics are more toxic when used intravenously [36] and should be given by this route only in acute emergencies the intramuscular or subcutaneous route should be employed as soon as possible thereafter *Bloodless phlebotomy* by tourniquets [46] if employed properly often is markedly effective in patients with pulmonary edema secondary to low output cardiac failure [51] Tourniquets having been

placed on three extremities sufficient pressure is exerted to occlude the veins but not the arteries Pressure is applied for fifteen to twenty minutes and then the tourniquets are released and rotated so that one extremity is always free of pressure If after forty five or sixty minutes no definite improvement occurs a phlebotomy should be attempted 500 to 800 cc of blood being withdrawn rapidly Withdrawal of less than 500 cc rarely produces the desired decrease in blood volume but if an adequate amount is withdrawn quickly a resultant fall in venous pressure and a rise in cardiac output can be expected This increase in output occurs because the heart is once again able to respond to stress *i e* the decreased venous return allows the overdistended ineffective cardiac muscle fibers to decrease in size and contract more forcefully within the limits of Starling s law of the heart The hemoglobin and hematocrit should be checked before phlebotomy is done and if the results obtained are markedly reduced from the normal values expected this procedure should not be considered On the contrary in pulmonary edema secondary to severe anemia with high output failure small transfusions of packed red blood cells may be life saving Actual phlebotomy should be done only in unequivocal cases of severe low output cardiac failure after other measures including bloodless phlebotomy with tourniquets have failed

Therapy of Congestive Heart Failure — After the acute attack of left ventricular failure is over the usual management of congestive heart failure is instituted Maintenance digitalization with Digoxin 1 to 3 tablets (25 to 75 mg) per day a low sodium diet and the judicious use of mercurial diuretics are the three cardinal features of long term therapy In addition *penicillin* (300 000 to 600 000 u daily) in a single dose should be administered in cases of moderate or severe heart failure to prevent secondary lung infections and Dicumarol should be given to prevent pulmonary infarction especially in patients who will be

bedridden for long periods of time [210] We believe that the immense value of these measures in congestive failure has been largely overlooked In our experience they have improved the course of apparently hopeless cases

Adequate restriction of salt can now be achieved while the patient enjoys a reasonably well balanced palatable diet [217] During the first week after the acute episode of heart failure a diet containing not more than 1 gram of sodium should be prescribed This can be accomplished by allowing only fresh fruits unsalted vegetables potatoes or rice salt free milk (prepared from lanolac powder) cottage cheese and salt free bread If the patient does well on this regimen small portions of meat and fresh water fish may be given The patient should be warned against eating the following foods

- Salt butter margarine or peanut butter
- Crackers cakes or pastries or any food made with baking soda
- Smoked or salt cured meats and fish ham bacon pork or corned beef
- Canned soups or vegetables
- Pretzels salted nuts potato chips candy beets celery lima beans spinach and sauerkraut
- Olives pickles catsup mustard or salad dressings

A salt substitute, *i e*, potassium chloride, may be used but most patients find this preparation bitter and unsatisfactory and prefer to use no salt at all Palatability may be increased by flavoring with pepper vinegar, garlic onions vanilla chocolate or cinnamon There should be no restriction placed upon the ingestion of water A fluid intake of between 2 and 3 liters per day seems to be the optimal intake for maximal sodium excretion [101, 119] Tea coffee fruit juices ginger ale lemonade salt free milk (lanolac) and occasionally small quantities of alcoholic beverages may be taken Frequent small meals are to be preferred to a few large ones and under no circumstances should a large meal be eaten within three to four hours before bedtime The

patient should be cautioned about the use of sodium bi carbonate or laxatives containing sodium

If a salt poor diet *i e* a diet containing between 1 and 2 grams of sodium daily, is followed carefully, many recurrences of attacks of acute pulmonary edema or severe congestive failure can be eliminated

Patients who are obese should be put on a fairly rigid reducing diet, and their weight should be kept at a minimum, if possible We have seen patients with severe cardiac disease and intractable heart failure who were greatly improved and well controlled after marked weight reduction Obese cardiacs are much more difficult to control than patients with normal weight Great stress, therefore should be placed upon weight reduction as an integral part of the therapy of congestive failure If these dietary precautions are observed patients may be able to continue for many months without any additional therapy If however the patient is unable to adhere to a proper salt poor diet or if his weight increases or edema develops while on such a diet mercurial diuretics should be used as often as is deemed necessary to keep the patient edema free and at a stable weight

Intramuscular Mercuhydrin in 1 to 2 cc doses has been used extensively, and is a safe effective mercurial which causes little pain at the site of injection It may be used subcutaneously or orally but its action is not wholly satisfactory when given by these routes

Thiomerin a mercurial which is given subcutaneously is effective and easy to administer,[15, 105] and we have used this drug with excellent results It is possible to teach patients to administer it to themselves, this greatly facilitates therapy and is as effective and safe as is insulin administration Since exercise increases sodium retention,[106] patients are instructed to stay in bed, if possible, for an hour or two following each injection In this way the maximum diuretic effect may be obtained Occasionally minor muscle

cramps occur two hours after the first dose of this medication but this discomfort disappears quickly

Oral mercurial diuretics may produce a satisfactory response in patients with congestive heart failure [57] but the use of these preparations has thus far been limited because of toxic reactions[59] and uncertain effects In patients who require two or more injections of a diuretic weekly it is often possible to eliminate one of the injections by using a longer acting oral mercurial (Mercuhydrin Mercuhydrin ascorbic acid Salyrgan or Mercuzanthin tablets) but usually the oral medication is not dependable enough to allow elimination of parenteral therapy Occasionally however a patient can be completely controlled by oral therapy A seventy one year old woman with chronic rheumatic heart disease with auricular fibrillation and with chronic heart failure was treated with Digoxin and mercurial injections for many years but because the mercurial injections proved painful they were discontinued An oral mercurial preparation (Salyrgan theophylline) was then given (2 tablets daily equivalent to approximately 1 5cc of the parenteral preparation) The dosage was reduced to 1 tablet daily after one year and the patient has been controlled satisfactorily on this medication for three years Certainly if an oral mercurial preparation that produces a satisfactory prolonged effect is found it will further simplify diuretic therapy

Occasionally *it is necessary* to use ammonium chloride 4 to 6 grams (60 to 90 gr) a day for two to three days prior to mercurial administration to potentiate the mercurial effect [33] Enteric coated tablets containing 1 gm each given in small and frequent doses are well tolerated but the drug should not be used continuously or in patients with marked renal impairment because of the possibility of inducing severe electrolyte disturbances *i e* hypopotassemia and acidosis Aminophyllin 0 5 gm (7½ gr) given intravenously increases renal blood flow and when used sixty

to ninety minutes after a mercurial diuretic may markedly increase the diuretic response [760]

Mercurials may be given safely in the presence of renal disease unless oliguria or azotemia is present but great care must be taken when administering mercurial diuretics to elderly persons to avoid excessive diuresis which may result in salt depletion dehydration and azotemia [52] For this reason it is wise to check the blood urea and the sodium or chloride levels from time to time When salt depletion occurs it is often attended by serious chemical derangements and symptoms [127, 63] the most important of which are extreme weakness apathy disorientation stupor coma and rarely death and while the daily injection of a mercurial often is dramatically effective in young persons it may be dangerous for the elderly patient For example

A seventy year old man who had been in congestive failure for four years had received a mercurial injection of 2 cc every week during that period and apparently had been well controlled until three weeks prior to admission He was admitted to the hospital for treatment of severe right and left heart failure The specific gravity of the urine was 1 020 the blood urea nitrogen 15 mg per cent the urea clearance 54 per cent in two hours and the phenol sulfonphthalein excretion 35 per cent After receiving 2 cc of Mercupurin by vein daily for ten days he had lost 17 pounds and all signs of congestive failure had disappeared The urea nitrogen had risen to 67 mgm per cent however and the patient had become disoriented lethargic and hyperpneic He began to vomit and developed a tremor of the hands Urinary output dropped to 85 cc in twenty four hours and the blood urea nitrogen rose to 109 with a blood chloride level of 679 mg per cent An intravenous drip of glucose in saline was given continuously but he died sixteen days after the last mercurial injection

For older people only 1 cc of Mercuhydrin should be given intramuscularly or 1 cc of Thiomerin subcutaneously not more than every other day after the acute attack of

heart failure has been controlled. In elderly men mercurials often aggravate prostatic disease causing acute retention and severe uremia.[219,254] In these cases mercurials should be given with even greater caution. It has been demonstrated that morphine, Demerol, quinidine, ephedrine and Adrenalin often decrease the effectiveness of the mercurial diuretics and these drugs should be avoided if possible while the mercurials are being given.[81,181] It may also be wise to reduce the dose of digitalis on the day that a mercurial diuretic is given in certain patients who are markedly edematous. If this is not done, mobilization of digitalis with the edema fluid[324] may result in the release of excessive amounts of digitalis and digitalis intoxication.[6,4] This complication of mercurial administration is not stressed but is probably much more common than previously suspected. We have seen several patients whose digitalis dosage had been well regulated and who developed signs of digitalis toxicity, *i.e.*, nausea, vomiting and ventricular premature beats *within twelve to twenty four hours after* the initial dose of a mercurial diuretic.

The judicious use of the mercurial diuretics with adequate salt restriction will usually control cardiac edema but in some refractory patients who are not able to stay on a salt poor diet and who are perpetually in chronic congestive failure, the use of *cation exchange resins* may be justified if given carefully under close supervision. These resins, which are substances capable of "binding" ingested sodium within the intestinal tract and excreting it in the stools,[6] are now available for general use. Hitherto their administration was restricted because a great many pills had to be ingested to produce effects and excessive amounts of potassium were eliminated along with other electrolytes, resulting in severe electrolyte disturbances. The new ammonium cation exchange resins are prepared in powder form, have added potassium and may not present this difficulty.[14] The safety of even these preparations has not been proven conclusively and they should be used cautiously and only

when close supervision of the patient is possible Blood electrolyte determinations should be done frequently at least for the first few weeks following the onset of therapy If hypopotassemia or rapid sodium depletion occurs the resin should be temporarily discontinued Our experience thus far has indicated that if administered carefully these substances are of value in the therapy of congestive failure especially in the cases where patients do not remain on an adequately controlled salt poor diet

In addition to the above measures the patient s activity should be carefully regulated Short periods of bed rest *should be taken if symptoms become severe or if heart* failure appears to be progressing

In heart failure secondary to hyperthyroidism pernicious anemia beri beri or surgical lesions the correction of the primary disease usually prevents its recurrence In cardiac failure secondary to tachycardia treatment of the latter condition should be instituted as outlined above

In patients with heart failure fluid may accumulate rapidly in the chest and cause acute distress In these cases immediate and repeated thoracenteses give remarkable relief and permit recovery Accumulation of fluid in the abdominal cavity also occurs but rarely does enough fluid collect to cause symptoms and require paracentesis This has been especially true in recent years since the use of good salt poor diets and adequate mercurial therapy became widespread Occasionally in patients with severe rheumatic heart disease and long standing congestive failure massive ascites will develop as a result of advanced cardiac cirrhosis we have seen patients who required frequent paracenteses in order to remain comfortable One of these was a thirty four year old female who was hospitalized because of chronic congestive heart failure, secondary to rheumatic heart disease Bilateral pleural effusions and ascites were found Despite adequate digitalization marked salt restriction and the frequent use of mercurial diuretics, little improvement was seen after one week of therapy A paracen

tesis was performed and 7 liters of fluid were removed A marked diuresis with dramatic clinical improvement occurred during the next four days Another paracentesis was done one week later thereafter the patient was adequately controlled and required no more tapping

Cardiac cirrhosis and liver disease in patients with chronic congestive failure is apparently much more common than previously suspected[25 14] and for this reason attention should be paid to the patient s intake of protein and vitamins Because these patients are often on a strict diet there is a tendency to overlook simple measures such as the use of protein and vitamin supplements that might postpone the occurrence of severe liver disease and ascites

During attacks of acute heart failure especially following coronary occlusion annoying and often serious symptoms may occur *e g* hiccup severe nausea vomiting and distention the latter symptoms occasionally result in death It is often of great importance to stop these symptoms quickly or prevent their occurrence

The excessive use of opiates should be avoided if possible and laxatives should be given freely to avoid constipation In the presence of continuous narcotic medication two and even three times the usual dose of the laxative may be necessary If therapy with laxatives such as Milk of Magnesia (30 to 60 cc) or Cascara sagrada (10 to 30 gr) is not successful occasional low gentle enemas may be used in conjunction with the above Cathartics that act upon the large bowel such as Cascara sagrada or rhubarb are not to be given however in patients receiving mercurials because of the possibility of inducing severe colitis

We have found intravenous pyridoxine (25 to 50 mg) of great value in treating the nausea and vomiting which complicates congestive failure Other measures that we have found of value in treating these symptoms include sedation restriction of foods and fluids sips of ginger ale Dramamine (100 mg every four hours if retained by mouth) and the elimination of the possible causative agents such as digi

talis morphine aminophyllin and ammonium chloride Certainly there is no indication for the forcing of cold milk and fruit juices as is done so often since these tend to increase nausea and the nurse or patient s family should be instructed specifically against this practice However milk which is boiled and then cooled to room temperature is usually well tolerated The fruit itself instead of the juice is also satisfactory

Intractable hiccup has been treated successfully with sedation amyl nitrite intravenous niacin inhalation of 5 to 7 per cent CO_2 and 90 per cent oxygen for a few minutes at a time ethyl chloride spray to the region over the diaphragm inhalation of ether or intramuscular quinidine gr 6 (4 gm) 3 times a day [23] A phrenicectomy or exeresis is occasionally required to stop a severe attack

Chapter

3

ANGINA PECTORIS CORONARY INSUFFICIENCY AND CORONARY OCCLUSION

TREATMENT

THE most common cardiac emergency is pain in the chest It is essential to exclude extracardiac causes of chest pain such as spondylitis herpes zoster pneumothorax pleurisy gallbladder disease peptic ulcer hiatus hernia and esophageal spasm *before deciding that the pain is cardiac in origin* This can usually be done by taking a careful history doing a complete physical examination and employing routine laboratory tests Once the cardiac origin of the pain has been established the type of the acute cardiac episode must be determined The character intensity and duration of the pain may help the physician to distinguish between attacks of coronary insufficiency including angina pectoris and coronary occlusion The pain in coronary occlusion is prolonged severe and unrelieved by nitrites whereas in coronary insufficiency particularly angina pectoris it is usually briefer and is relieved by nitroglycerin Occasionally however coronary occlusion is associated with only mild pain and no other symptoms whereas coronary insufficiency may cause severe pain The entire clinical picture and the electrocardiographic changes should be considered before a definite diagnosis is made and occasionally it can be made only after observation for several days Any patient therefore who experiences sudden precordial pain diagnosed to be cardiac in origin should be put to bed and

treated as a case of potential infarction although many of these cases do not progress to a coronary occlusion with infarction

Angina pectoris, precipitated by effort exposure to cold, excitement or following large meals is a good example of temporary coronary insufficiency with ischemia of the sub endocardial layer but no myocardial necrosis The ordinary episode of angina pectoris is quickly relieved by rest or nitroglycerin a hypodermic tablet gr 1/200 to 1 150 being given under the tongue The patient should be instructed to take the nitroglycerin immediately if the pain *is severe or if a specific activity such as eating or walking in* cold weather is known to induce anginal pain the drug should be taken prior to this activity For example many patients experience pain only in the morning on the way to the subway or train a nitroglycerin tablet under the tongue just before leaving the house often prevents the attack This drug is safe and extremely effective and should be taken as often as necessary If nitroglycerin is not used and the pain persists the patient becomes frightened and anxious this emotional state serves to perpetuate the attacks The establishment of a vicious circle of pain anxiety and more pain makes therapy extremely difficult but liberal use of nitroglycerin and sedation often interrupts this seemingly endless chain There is apparently no danger in giving nitroglycerin as frequently as every hour Occasionally however it produces flushes intense throbbing and severe headaches or even syncope and in these instances it should be used with caution For example A forty eight year old waiter suddenly experienced substernal pain and was given gr 1 150 of nitroglycerin Severe throbbing in the head which lasted for several hours profuse perspiration and ' faintness occurred but the pain was not relieved During another attack of precordial pain several days later the patient was prevailed upon to test the effect of nitroglycerin in a minute dose of gr 1/400 but, again the same symptoms were produced Of course,

if several doses of nitroglycerin do not control the attack or if the pain lasts for more than twenty to thirty minutes the medication should be stopped and the patient put to bed and observed carefully for other signs of coronary occlusion Treatment should then be instituted as described below

Whiskey is occasionally effective in the treatment of angina and sometimes a patient prefers to take small sips of whiskey instead of nitroglycerin for the attacks A few cases do not seem to respond to either nitroglycerin whiskey or to adequate sedation These patients may continue to have frequent severe attacks of angina which at first appear only after exercise but later recur frequently at rest *i e status anginosus* In many instances this condition heralds the onset of a coronary occlusion When status anginosus occurs therefore the patient should be put to bed under adequate sedation and carefully observed for several days The period of bed rest and sedation may alleviate the pain and allow the patient to resume at least mild activity upon arising In those patients who do not show evidence of occlusion but who continue to have severe bouts of anginal pain other methods of treatment have been used and are being evaluated

Many observers have used antithyroid drugs [21,23] and even thyroidectomy [30] for the treatment of severe angina hoping thereby to reduce cardiac work by lowering body metabolism Good results have been reported in some cases after all other treatment has failed but these procedures do not appear warranted because of drug toxicity or surgical morbidity and mortality More recently radioactive iodine I_{131} has been employed to produce a hypothyroid state in angina patients with some success in intractable cases [31] This type of treatment is usually not required in cases of angina pectoris and we have rarely resorted to the use of anti thyroid therapy in euthyroid patients Occasionally when hyperthyroidism is present or aggravates a pre existing anginal syndrome, correction of

the hypermetabolic state by radioactive iodine may completely relieve the anginal attacks In older persons there may be no obvious signs of hyperthyroidism and only after a complete study can the condition be diagnosed

A F was a sixty seven year old Italian housewife who entered the hospital because of severe recurrent episodes of substernal pain which began suddenly two weeks before Her blood pressure was 160 80 her rhythm was regular and a grade ++ apical systolic murmur was heard The ECG showed deeply inverted T waves in the standard and precordial leads a diagnosis of coronary insufficiency was made In spite of complete bed rest and large doses of sedatives she continued to experience frequent attacks of pain which were only partially relieved by nitroglycerin During the course of a thorough study her basal metabolic rate was found to be +40 per cent and she excreted only 10 per cent of a test dose of I_{131} indicating hyperthyroidism She was given a therapeutic dose of I_{131} and Lugol s solution and after several weeks her anginal seizures began to subside and finally disappeared completely

Papaverine has been advocated for the treatment of angina but careful studies have shown that in doses up to 800 mg by mouth daily it has no significant effect upon the anginal syndrome [234] The original claims that Alpha Tocopherol (Vitamin E) was of value in the treatment of angina pectoris have also been refuted [0] We have used both of these medications with poor results

Khellin an extract from the seeds of an Egyptian plant is the most recent drug to be used in the treatment of angina It dilates the coronary arteries when given intramuscularly in doses of 100 mg or orally in 40 to 60 mg doses 3 times daily Some favorable reports on the efficacy of Khellin[4] have not been confirmed [0] Our own experience has shown it to be of little value thus far in most of the patients who have received it gastro intestinal symptoms such as nausea and vomiting have developed

For many years various *surgical procedures have been*

employed to relieve pain in cases of angina that responded poorly to nitroglycerin that recurred frequently and that required liberal use of narcotics for their control Paravertebral injection of the upper five thoracic sympathetic ganglia and their rami communicantes with alcohol [9] has occasionally provided relief for patients[147] who were not amenable to other types of treatment This method or the injection of the stellate ganglion alone with ammonium sulfate [238] appears to be achieving some satisfactory results in experienced hands More recently good results have been reported from the use of various ganglionic blocking agents such as tetra-ethyl ammonium chloride especially in patients with status anginosus [8] There does not appear to be any justification at the present time for the use of surgical procedures that attempt to increase collateral coronary blood flow such as omental grafts,[54] implantation of irritating substances into the pericardial cavity [52] or the anastomosis of the aorta to the coronary sinus as has been advocated [15]

We have been most successful in improving the condition of our anginal patients by the frequent unhesitating use of nitroglycerin by reassurance and by the judicious use of sedatives and narcotics We believe that most patients should be treated by these means and that the use of other drugs or surgical procedures is only rarely justified In only one instance has it been necessary for us to resort to surgical therapy in a case of angina pectoris during the past four years In this case an upper thoracic sympathectomy was performed but the result was unsatisfactory The patient continued to have precordial pain and in addition developed severe postoperative neuralgia

Coronary Insufficiency —In recent years many acute coronary episodes have been observed which simulate coronary occlusion at their onset but do not resemble it electrocardiographically and in their subsequent clinical course [176, 177] In these cases the ECG may show only RS-T depressions and/or T wave inversion which usually appear

soon after the attack although, in some cases, they may not become evident for several days or a week (Fig 7) Fever, leucocytosis, or elevated sedimentation rate may also be present, although, in the milder cases where myocardial ischemia without necrosis occurs, these findings are usually absent

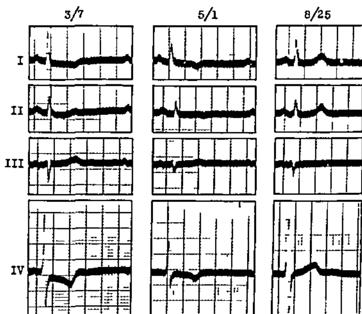


Fig 7 —N G m 53 Acute coronary insufficiency following physical exertion (Master and Jaffe courtesy of Postgraduate Medicine)

When attacks of precordial pain occur spontaneously, at rest, it is often extremely difficult to distinguish between those that are caused by simple coronary insufficiency and those that will progress to coronary thrombosis For example·

Mr. I F, a storekeeper, forty-four-years old, had been observed over a number of years because of numerous functional complaints *Physical examination, blood pressure,*

and ECG were normal One day while at work he experienced severe substernal pain which lasted one hour and closely resembled the pain of a coronary occlusion At this time he was not in shock his heart sounds were regular at a rate of 76 and of good quality He showed diphasic T waves in leads I II and V_{456} but his temperature was not elevated and there was no leucocytosis He remained asymptomatic but on the fourth day his sedimentation rate was elevated to 30 mm and his electrocardiogram showed inverted T waves in all leads without the presence of Q waves in any lead The T waves gradually became upright and in four weeks the electrocardiogram was again normal He remained well during the next three years and his ECG remained unchanged after exercise

A J a sixty three year-old automobile salesman with a history of bronchial asthma for many years complained of sudden severe pain across the upper back radiating anteriorly He perspired freely his blood pressure was 130 80 a drop of 30 points from his usual systolic pressure The pain lasted forty five minutes and was relieved by 100 mg of Demerol subcutaneously Despite a normal ECG he was suspected of having suffered an acute coronary occlusion After four days he was permitted out of bed in a chair but he again began to experience frequent severe pain in the upper back during the day and particularly at night Dicumarol therapy was begun and the patient received oxygen by mask aminophyllin suppositories Demerol 100 mg q 4 h and sedatives The pain increased in severity and intensity but the patient s temperature white count and sedimentation rate remained normal On the eleventh day the ECG showed slight inversion of the T wave in lead I and V_{2-5} but no other changes The T wave inversion gradually increased during the following two weeks and then receded four weeks after the acute episode the ECG had returned to normal

In these cases coronary insufficiency with subendocardial ischemia without actual coronary occlusion clinically

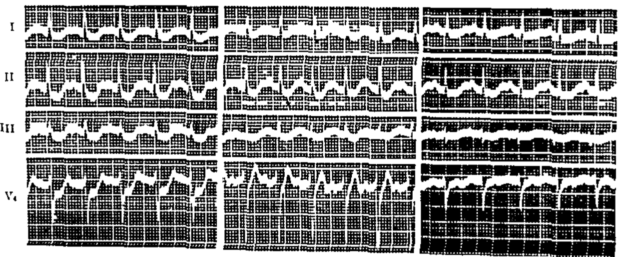


FIG 8 —P I m 56 Acute coronary insufficiency in duodenal ulcer with profuse hemorrhage Necropsy showed subendocardial necrosis *A* Marked RS–T depression in all leads *B* RS–T depression less marked *C*—Further improvement (Master Dack Horn Freedman and Field courtesy of Circulation)

resembled a coronary thrombosis and in the second case anticoagulant therapy was started before the actual diagnosis of coronary insufficiency was made

In the more severe cases of coronary insufficiency there may be mild or moderate fever leucocytosis and alteration of the sedimentation rate and longer lasting ECG changes These may occur following operative procedures massive hemorrhage[175] or tachycardias Coronary insufficiency complicates gastrointestinal hemorrhage in many cases especially when there is a marked fall in blood pressure (Fig 8) For this reason we do not hesitate to give large amounts of blood to a patient who is actively bleeding especially if he has coronary artery disease All attempts should be made in these cases to keep the blood volume and blood pressure as close to normal levels as possible Frequent cardiograms should be taken if acute coronary insufficiency occurs despite all efforts to prevent it the patient should be kept in bed until the cardiographic abnormalities disappear These cases usually recover in the fatal cases necrosis of the subendocardial layer with evidence of coronary sclerosis is found but there is no evidence of an acute thrombosis[87]

It is important to distinguish coronary insufficiency from coronary occlusion in the former anticoagulant therapy is not usually necessary the period of bed rest required is shorter and the prognosis is usually much better In cases secondary to postoperative shock massive hemorrhage severe diarrhea or dehydration treatment of the primary condition usually results in complete recovery from the cardiac difficulty

Efforts to prevent attacks of severe coronary insufficiency should be made in all patients with any evidence of coronary disease They should be advised not to indulge in strenuous activities they should also be warned against excessive eating exposure to extreme change in temperature especially cold frequent sexual intercourse emotional upsets and excessive smoking All attempts to prevent a fall in blood

pressure should be made especially when such patients are subjected to surgery Spinal anesthesia may be used but care should be taken to prevent the blood pressure from falling below 100 mm Hg If a rapid fall in blood pressure does occur during surgery due to the anesthesia or to massive hemorrhage blood should be given freely If heart failure is induced by excessive fluid it may be quickly treated by digitalization the use of mercurials and the other measures previously described It is far better to risk the occurrence of congestive heart failure which can be treated later than to allow a patient to lapse into irreversible shock by withholding transfusions merely because the patient has heart disease

In diabetic patients with coronary artery disease who are receiving insulin it is important to prevent episodes of hypoglycemia it may be better to allow them to spill small amounts of sugar than to attempt to attain perfect sugar control since hypoglycemic reactions often precipitate episodes of coronary insufficiency *If the above precautions are followed many cases of this syndrome will be prevented*

Coronary Occlusion —The pain experienced in coronary occlusion usually begins suddenly at rest during sleep or in the course of routine activity It is usually precordial or substernal but may be situated only in the back of the chest or neck or in the arms Instead of pain the patient may complain of severe burning sensations or feelings of tightness nausea vomiting and profuse perspiration Varying degrees of shock may be present The sudden onset of paroxysmal tachycardia or pulmonary edema is occasionally the first evidence of an acute coronary occlusion Examination may reveal gallop rhythm embryocardia (*tic tac rhythm*) or congestion of the lungs The blood pressure may be normal at first or it may drop precipitously

The clinical and electrocardiographic manifestations of coronary occlusion depend upon the pathological changes that occur in the myocardium *In over half the cases the*

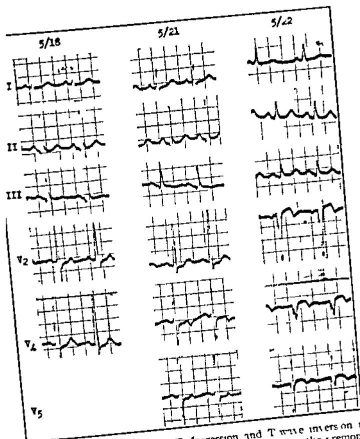


FIG 9 — N H m 45 RS–T depression and T wave inversion in the chest leads indicating coronary insufficiency during the premonitory phase of coronary occlusion (5 18 21) Following the acute attack RS–T elevation and Q waves appeared in leads V_2 and V_4 (5 22) indicating anterior infarction (Jaffe Courtesy of J Mt Sinai Hosp)

process of thrombosis is spontaneously initiated by a sub intimal hemorrhage which leads to secondary changes in the intima or may completely occlude the vessel lumen[128] This process of thrombus formation may take days or weeks During this period the patient may experience severe episodes of angina or recurrent pain at rest the so called premonitory phase of an occlusion[129] (Fig 9) The electrocardiogram may show RS T depressions and T wave inversions indicating coronary insufficiency with myo

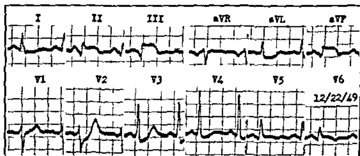


Fig 10 J S m 58 Acute coronary occlusion with postero lateral infarction (Master and Jaffe courtesy of Postgraduate Medicine)

cardial ischemia These changes are replaced by Q waves and ST segment elevations however as soon as the occlusion has become complete (Fig 10) A Q wave usually appears in one or more leads depending upon the site of the infarction and produces a characteristic pattern of Q and RS T elevation During the following days and weeks serial tracings reveal a progressive return of the RS T segment to normal and an inversion of the T wave resulting in a Q inverted T pattern which may persist for months or years Fever appears on the second or third day The sedimentation rate becomes prolonged by the fourth day, often to 50 mm or more In a through and through infarction the subpericardial region is involved and a pericardial

reaction[246] and friction rub may result Since the infarct reaches the intima mural thrombi may form and peripheral embolization occur [112]

As we have mentioned previously it is oftentimes extremely difficult to differentiate between cases of simple coronary insufficiency and those which represent the premonitory phase of coronary occlusion Both types therefore should be put to bed and observed before a diagnosis is made In many cases of course where Q waves appear within eight to twenty four hours following the onset of pain a definite diagnosis can be established quickly It is also extremely difficult in many instances to distinguish between a coronary occlusion and an attack of acute pericarditis Some of the features that differentiate these conditions from one another and from coronary insufficiency are summarized in Table II

When electrocardiographic and clinical evidence of a coronary occlusion with myocardial infarction is present therapy should be begun immediately Pain should be alleviated before other measures are instituted If it is mild Demerol 50 to 100 mg or codeine 03 to 06 gm (gr ½ to 1) should be given orally and will usually suffice In more severe cases however morphine 15 mg (gr ¼) subcutaneously will be effective but it may occasionally be necessary to give morphine 10 to 15 mg (gr 1 6 to 1 4) intravenously These doses may be repeated in twenty to thirty minutes The possibility of inducing distention obstipation and nausea and vomiting with morphine is great and the dose used should be no larger than is actually required The morphine should be discontinued as soon as any of the above symptoms appear because distention and vomiting may turn an otherwise benign course into a stormy one We have used Dilaudid extensively and especially in cases where morphine induced vomiting we have found it to be a reliable analgesic and to be without the unpleasant and dangerous side effects of morphine Two and one half to 5 mg (1 24 to 1 12 gr) usually produce

quick relief of pain We have had some experience with Pantopon and Metapon, opiate derivatives that produce few toxic reactions and have found these drugs also to be satisfactory Nitroglycerin should not be used for the control of pain once a diagnosis of coronary occlusion has been made since it may further lower blood pressure and decrease coronary blood flow

TABLE II

	Pain	Severe Shock	Gallop Rhythm	Pericardial Rub	Heart Failure	Arrhythmias	Di p Bloo Pressu
Coronary Insufficiency a) Mild Angina Pectoris	Few seconds to 10–15 minutes	Absent	Usually absent	Absent	Absent	Absent	Absent
b) More Severe—Spontaneous or Secondary	May be more prolonged	May be precipitating factor e.g hemorrhage	May occur	Absent	Uncommon but may complicate cases due to tachycardia or hemorrhage	Uncommon	Moderate ... sod pr... itated ... h m rrh... r tachy... dia
Coronary Occlusion	Usually severe longer than 15 minutes may rarely have no pain	Present in about 10 per cent of cases	Commonly occurs	Common	Common	Both auricular and ventricular arrhythmias may occur	Often mark...
Pericarditis	Mild rarely severe	Rare	Rare	Usually present	Uncommon	Auricular flutter and fibrillation	Rare may ... sult (ca... ac tamp... ade occur...

Oxygen should be used in all cases where the pain persists after morphine has been given A tent should be employed where possible, especially when prolonged oxygen therapy is anticipated because the use of a mask or nasal catheter often increases the patient's anxiety Occasionally, intravenous aminophyllin, 0 5 gm, (7 5 gr) is of value In rare instances, sodium amytal, 0 5 gm (7 5 gr) by vein, has been effective when all other measures have failed

The patient should be put to bed in a quiet room if possible, and all distracting influences such as telephone and mail, should be banned. The importance of obtaining prolonged physical and mental rest should be explained to the patient. Visitors should be limited to members of the immediate family for the first two to four days.

The most serious complication of coronary occlusion is

TABLE II

Fever	Leucocytosis	Elevated Sedimentation Rate	ECG RS-T El., RS-T Dep., Q-Waves	Pathology	Precipitating Factor	Treatment
[Ab]sent	Absent	Rare	RS-T El. Absent; RS-T Dep. Transitory; Q-Waves Absent	No pathological changes	Emotion, exercise, cold, large meals, etc.	Nitroglyc[erine]
[Ma]y be present	May be present 11,000–15,000	Moderate	RS-T El. Rare; RS-T Dep. Common; Q-Waves Absent	Subendocardial necrosis	1. Tachycardia, hemorrhage, trauma; 2. Spontaneous	Treat etiological factor
[Us]ually present after first 36 hours	Usually present after 36 hours, may be over 15,000	Usually after 36 hours	RS-T El. Common; RS-T Dep. Reciprocal; Q-Waves Common	Through and through infarction of myocardium	Usually no definite precipitating factor	Symptomatic, anticoagulants, rest
[Co]mmon	Depending upon etiology. If viral, leucopenia is present	Common	RS-T El. Common; RS-T Dep. Rare; Q-Waves Absent	No myocardial damage	Unknown, rheumatic fever, virus, bacterial, traumatic	Treat primary disease

shock, which is due to a sudden failure of the heart to pump blood to the tissues, *i.e.*, a fall in cardiac output[175] and is not due to blood loss or to a decrease in venous return. This so called "cardiogenic shock" results from myocardial weakness caused by the infarct and from certain poorly understood reflexes. Theoretically, then, the use of transfusions or intravenous fluids is not indicated in these cases and in fact may lead to a further increase in venous pressure

and congestive heart failure Actually however the danger of inducing failure thereby is not great as has been shown by several recent studies [73 2-3] Although this danger has also not proven significant in our own experience failure only occurring in a small percentage of cases the administration of intravenous fluids has reversed the shock process and changed the eventual outcome in only a few cases Intra arterial transfusions may prove more useful although sufficient data have not yet been accumulated with this method to judge its value The patient in shock should be placed in a moderate Trendelenburg position and should *be given oxygen If he complains of severe pain morphine* Dilaudid or Pantopon should be given as required Small doses of neosynephrine 3 to 5 mg may be given intramuscularly and may be repeated every two to three hours This drug raises blood pressure without affecting the myocardium adversely and is therefore safer than epinephrine If neosynephrine is not available Paredrine 5 to 10 mg (1 12 to 1 6 gr) or norepinephrine may be used If a patient remains in shock despite these measures for more than an hour or two or if only minimal improvement occurs small transfusions should be given If blood is not available plasma or hypertonic glucose solutions should be administered until blood is obtained These measures are employed in an effort to prevent irreversible shock and death although they are effective in only a small percentage of cases One of us (A M M) has used ACTH and cortisone in this condition in a few instances but the results thus far have been poor Usually the patient will recover from the shock state within a few hours or will expire Occasionally patients have remained in shock following a coronary occlusion for as long as 8 to 12 hours and have recovered but this is not the usual course of events If pain is promptly relieved oxygen given and the other simple measures outlined above are carefully followed the percentage of patients who recover will be as high as can be expected from any form of treatment in this condition

Clinical evidence of mild left ventricular failure is very commonly observed early in the course of coronary occlusion, a few râles are found in the chest and slight congestion of the lung fields is seen on x ray As a rule, this type of failure requires no specific therapy or responds promptly to moderate salt restriction and to an injection of a mercurial diuretic Occasionally, however, acute pulmonary edema is the first sign of a coronary occlusion or severe heart failure develops within several days after the occlusion The treatment of heart failure in the course of coronary occlusion and in the course of chronic heart disease is the same, except for the use of digitalis, concerning this, there is a difference of opinion Many physicians believe that digitalis should be administered routinely in congestive failure occurring during a coronary occlusion, just as if the latter were not present [86] There are reasons however, for exercising caution in the use of this drug the myocardium in the region of the infarct is more irritable, and digitalis may increase the irritability inducing premature beats, ventricular tachycardia, and even ventricular fibrillation Many of the episodes of ventricular tachycardia in coronary occlusion have occured in patients receiving digitalis and the association is probably not entirely coincidental In the course of a coronary occlusion, therefore, we first employ other measures for combatting the associated congestive failure In pulmonary edema we give oxygen morphine and if necessary intravenous aminophyllin and a mercurial diuretic If relief is not obtained however, we do not hesitate to inject Strophanthin K (or Ouabain, Strophanthin G) intravenously, the initial dose being 0 25 mg This dose may be repeated within one hour, but the drug should not be used if digitalis had been given within the previous forty-eight hours In unusual instances, if it is felt that digitalization is absolutely necessary, doses of 1 mg may be given even if there is a history of recent digitalis intake In less acute failure we employ digitalis only if mercurial diuretics and a salt poor diet do not produce improvement In acute

coronary occlusion if we must digitalize the patient we do it somewhat more slowly than usually but otherwise follow the plan outlined in Chapter 1 p 21

Paroxysmal arrhythmias of all types frequently occur in coronary occlusion Premature beats auricular fibrillation and flutter and supraventricular tachycardia are most commonly encountered They usually do not alter the prognosis because as a rule they are of brief duration and remit spontaneously For this reason it is not necessary to begin treatment of these arrhythmias for several hours except in patients with heart failure or shock

M W a retired business man of seventy nine developed a coronary occlusion with anterior infarction and did well for two days On the third day his heart rate suddenly increased to 180 The ECG showed a supraventricular tachycardia with bundle branch block The arrhythmia was remittent for several hours and then was constant for four hours the patient became quite uncomfortable and dyspneic It was decided therefore to administer intramuscular quinidine 0 4 gm (6 grains) but by the time this had been drawn into the syringe the arrhythmia had ceased and did not recur

Treatment was withheld in this patient whose tachycardia remitted spontaneously In cases however in which the tachycardia persists all available measures should be employed to stop it Also in all cases with heart failure and shock treatment of the tachycardia should be instituted immediately The arrhythmias are treated as described in Chapter 1 Sedatives should be used liberally in these patients because the anxiety resulting from the rapid heart rate superimposed on the recent severe chest pain may prove difficult to manage

Ventricular tachycardia in acute coronary occlusion responds to Pronestyl and quinidine and these drugs should be given as outlined above (Chapter 1 p 35) A particularly difficult case was recently treated successfully with Pronestyl

M G a sixty year old tailor was admitted to the hospital because of melena and hematemesis of several days duration caused by a duodenal ulcer For several months he had experienced pain in the left arm on effort this symptom increased following the onset of bleeding Physical examination revealed a blood pressure of 114/65 and was negative The electrocardiogram however showed the pattern of an acute coronary occlusion with an anterior wall infarction and regular sinus rhythm The course of the disease was uneventful until the twelfth day when the patient became dyspneic and perspired profusely His heart rate was 190 and the rhythm was slightly irregular The electrocardiogram showed a ventricular tachycardia The patient received morphine intravenous quinidine 0 65 gm (10 grains) on two occasions intravenous magnesium sulfate and intramuscular quinine without effect He developed congestive failure within twenty four hours and was given Mercuhydrin and Digoxin 1 5 mg by mouth with some improvement in the failure He then received quinidine 0 4 gm (6 grains) every 2 hours by mouth The rate of the ventricular tachycardia ranged between 130 and 140 On the third day of the arrhythmia he was given 1 gm (15 grains) of procaine amide (Pronestyl) intravenously in fifteen minutes During the injection a few sinus beats appeared but the tachycardia recurred a short time after the drug was discontinued The next day 2¼ gm of Pronestyl were injected intravenously in thirty five minutes and numerous sinus beats appeared Within two hours regular sinus rhythm appeared and the patient made an uneventful recovery

Many years ago it was suggested[154] that quinidine be routinely administered early in coronary occlusion to prevent ventricular tachycardia We do no subscribe to this view because in our experience with 1000 cases of coronary occlusion who did not receive digitalis ventricular tachycardia occurred in only 0 5 per cent [174] It therefore is not wise to employ a drug with potential toxic effects such as quinidine in an attempt to prevent a complication which is so infre

quent Similarly we do not administer quinidine for premature beats unless they are numerous or arise in several foci Now that Pronestyl a relatively harmless but effective drug is available it may be given as soon as a significant number of ventricular premature beats appear but it too should not be used prophylactically in all cases of coronary occlusion to avoid ventricular tachycardia

Varying degrees of *P R* interval prolongation and evidence of partial or complete heart block may occur [75] following a coronary occlusion but these findings usually disappear spontaneously and are of no clinical importance

Anticoagulant Therapy —The major advance in the treatment of coronary occlusion during the past decade has been the introduction of anticoagulant therapy to prevent pulmonary and peripheral emboli The frequency of this complication in coronary occlusion has been reported to vary between 45 per cent [2] and 60 per cent[9] or more in pathological material although death may have occurred from other causes in many of these cases Anticoagulant therapy has been shown to reduce the frequency of embolic phenomena and of death in coronary occlusion by about 40 to 50 per cent [2] It is our distinct impression that many severe cases of coronary occlusion particularly those with congestive failure who have survived would have succumbed without the aid of anticoagulant therapy and we believe that this therapy should be used routinely in coronary *occlusion if adequate laboratory facilities for performing* prothrombin determinations are available

Pulmonary emboli most frequently originate in the lower extremities and lodge in the lower lobes [68] It is possible that a small percentage arise in the right auricle in the presence of auricular fibrillation or prolonged congestive failure The symptoms of pulmonary embolism are very variable There may be severe chest pain dyspnea and collapse simulating coronary occlusion or merely cough fever or tachycardia (Table III) Electrocardiographic abnormalities are present in half the cases the

TABLE III

	Pain	Dyspnea	Severe Shock	Hemoptysis	Gallop Rhythm	Pericardial Rub	Fever	ECG	X Ray
Pulmonary Embolism	often absent not typical	often severe	common in cases with large infarcts	present in ⅓ of cases	usually absent	absent	may be high	a) RS-T depression T wave inversion Coronary Insufficiency b) S Q3 pattern	Hazy patch in lower lobe may be present
Coronary Occlusion	usually severe substernal precordial left arm	often mild or absent	occurs in 5 10% of cases	absent unless pulmonary edema present	common	may be present in about 20% of cases	usually not over 101° 102°	RS-T elevation & Q waves Serial changes	may show minimal congestion or be completely normal

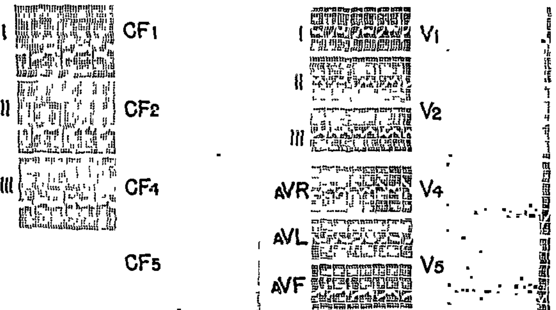


FIG. 11.—E.V. f. 40. Acute pulmonary embolism fifteen days following pelvic operation. Death nine hours later. Necropsy revealed bilateral pulmonary embolism of right main and left lower lobe branches. The posterior papillary muscle of the left ventricle showed *focal necrosis*. *AM*—Record taken soon after onset showed RS-T depressions. *PM*—Record several hours later showed prominent S wave in leads I and II and RS-T rounding in lead III. (Dack, Master, Horn, Grishman and Field. Courtesy of Am. J. Med.)

commonest single pattern being that of coronary insufficiency i e RS T depression and T wave inversion[53] (Fig 11) Other patterns are those of acute cor pulmonale i e a large S_1 and Q_3 with RT_3 elevation and occasionally of right bundle branch block Pulmonary emboli usually do not occur until after the first week of the attack most commonly during the second week About one third of the patients with pulmonary embolism expire within a few minutes or hours despite all treatment but if a patient survives for more than three to four hours he probably will recover[255] *In many cases it is extremely difficult to distinguish between pulmonary embolization and fresh myocardial infarction* The following points will be helpful in making a differential diagnosis

Although fever tachycardia and chest pain may be present in both conditions and cough may occasionally occur in cases with coronary occlusion and mild congestive heart failure the presence of cough and hemoptysis favors the diagnosis of pulmonary embolism Calf tenderness or a positive Homan s sign [1] indicates the presence of a thrombotic process in a lower extremity and therefore possible pulmonary embolization A loud pulmonic second sound not previously heard may indicate pulmonary hypertension secondary to infarction and the findings of a hazy patch of infiltration on chest *x* ray may further suggest the diagnosis of an embolic episode Electrocardiographic changes as described above may be present in pulmonary infarction but characteristic Q waves are absent All of these findings may help to distinguish between the two conditions Occasionally a pulmonary infarction occurs during the course of a coronary occlusion without the appearance of chest pain cough and electrocardiographic findings In these instances a persistently high fever or persistent leucocytosis may suggest the diagnosis

Arterial emboli to the brain the abdominal viscera and the peripheral vessels arise in mural thrombi in the left ventricle at the site of the infarction Since these thrombi

probably begin to form very soon after the onset of the attack anticoagulant therapy should be started as early as possible

The two anticoagulant drugs commonly used are *heparin* and Dicumarol The former is given by injection and its effect is evident within an hour or two[10] Dicumarol is administered orally produces a definite effect in thirty six to forty-eight hours and a maximum effect in seventy two to ninety six hours We believe that its action begins in twenty four hours in many cases although the ordinary laboratory tests may not detect this early effect Heparin alone may be used throughout the attack but because its injection is often painful and because it is expensive most physicians give both heparin and Dicumarol during the first two days of the attack and thereafter maintain the patient on Dicumarol In this way an immediate anti coagulant effect is produced by the heparin which after the third day is maintained by the Dicumarol

As soon as a diagnosis of coronary occlusion is made the patient receives 100 mg of heparin subcutaneously or 400 mg of Depo heparin intramuscularly as well as 300 mg of Dicumarol orally A control prothrombin time is taken When given subcutaneously the dose of heparin is repeated every eight hours but when the intramuscular preparation is used a second dose need not be given for twenty four hours In many cases we have not employed more than one dose of intramuscular heparin and it is our impression that the results obtained are just as satisfactory as when repeated doses are given There is usually no need to use intravenous heparin In the above dosage heparin usually prolongs the clotting time to approximately double the control value *i e* from fifteen to twenty five minutes (Lee White method) and there is ordinarily no danger of hemorrhage The clotting time should be determined immediately before in order to obtain a pre treatment level and two hours after the first dose of heparin in order to discover patients who are sensitive to it Thereafter this determina

tion is unnecessary If hemorrhage does occur in the course of heparinization a 1 to 2 per cent solution of protamine sulfate is given by vein at a dose of 1 mg of protamine per mg of heparin being administered After the initial dose of 300 mg of Dicumarol a dose of 200 mg is given on the second day The administration of Dicumarol requires much closer supervision than does heparin since there is a tremendous variation in the response to it from person to person and in the same person from day to day Even after some stabilization of dosage appears to have been achieved further administration of the drug produces unpredictable effects in some cases Therefore during the first two weeks of treatment the prothrombin time should be determined daily before the next dose of Dicumarol is given In the majority of patients the dosage of 300 mg followed by 200 mg is quite safe but occasionally prolongation of the prothrombin time to fifty or more seconds occurs within three or four days and profuse hemorrhage results For example

L W a woman of twenty eight with chronic rheumatic valvular disease mitral stenosis congestive failure and auricular fibrillation experienced many episodes of peripheral embolization Following a recent one she received 300 mg of Dicumarol on the first day and 200 mg on the second Her prothrombin time was thirty six seconds on the third day and no Dicumarol was given on that day or on the next On the fourth day diffuse ecchymoses appeared over the entire body and her prothrombin time was ninety four seconds She received 2 doses of 75 mg of intravenous vitamin K within five hours and the prothrombin time fell to normal limits within eight hours An uneventful recovery followed

M R a garage owner of fifty six developed a coronary occlusion He received 300 mg of Dicumarol on the first day and 200 mg on the second His prothrombin time rose to fifty two seconds and did not fall during the following two weeks although he received no more of the drug

These cases illustrate the extreme variability of response that may occur following Dicumarol therapy

In Dicumarol therapy the prothrombin time should be maintained at about 2 to $2\frac{1}{2}$ times the control time i e between twenty five and thirty five seconds In most patients this result is achieved with a daily dose of 50 to 150 mg Urinalyses should be done every two to three days on patients receiving Dicumarol if a significant number of red cells are found in the urine the dosage should be reduced or the drug temporarily stopped If the prothrombin time is prolonged to sixty seconds or more or if bleeding occurs the drug should also be stopped and the pateint given intravenous vitamin K Hykinone (vitamin K) 72 mg is administered intravenously and this dose is repeated in four hours if indicated Usually the prothrombin time will be reduced towards normal levels within four to eight hours after 75 to 150 mg of vitamin K have been given In more severe cases frequent transfusions of fresh blood are invaluable Recently the use of vitamin K oxide has been *advocated by some observers who have found it to be more* efficacious than ordinary vitamin K preparations[189] in the prevention of serious hemorrhage

Because of the danger of hemorrhage Dicumarol or any other anticoagulant substance should not be administered to patients with a history of hepatic gastric or renal disease or with a hemorrhagic tendency

Occasionally a patient will develop hemorrhage while on anticoagulant therapy despite the fact that his prothrombin time has been reported to be within safe limits A good example of this is seen in the case of a fifty eight year old hotel proprietor who developed a coronary occlusion with anterior infarction and was given 300 mg and 200 mg of Dicumarol on the first two days On the fourth day his prothrombin time was twenty seconds but he passed a large tarry stool and his hemoglobin fell to 8 6 gm He was given a small transfusion slowly and recovered quickly

Anticoagulant therapy should be maintained throughout

the period of bed rest and until the patient has become ambulatory In no instance should Dicumarol therapy be used in an institution that is not equipped to do reliable prothrombin determinations Despite the reliability of a laboratory however mistakes are occasionally made in determining the prothrombin time and in all cases the clinical picture should be considered more important than the chemical findings when treatment is ordered It should be remembered that prothrombin times are not accurate when performed on patients who are receiving heparin in addition to Dicumarol Although heparin primarily effects clotting time it may significantly prolong the prothrombin time For this reason and because a Dicumarol effect is usually not noted for 48 hours it is useless to do prothrombin determinations during the first two days of treatment when the plan of therapy outlined above is followed Some physicians continue anticoagulant therapy indefinitely in order to prevent recurrent coronary thromboses[8] We have had no experience with this type of therapy but we do not believe that it has proven to be of value thus far

Anticoagulant therapy has thus far been employed only in large medical centers hospitals in the larger cities and well equipped medical clinics facilities for prothrombin time determinations being limited or unavailable in out lying rural areas and in small hospitals Recent investiga tions of a bedside method of doing these determinations have demonstrated however that a simple capillary method utilizing rabbit brain thromboplastin yields results which although not so accurate as those obtained by more elab orate techniques are accurate enough for all practical pur poses[166] Approximately 700 patients have now been followed with this method and results appear to indicate that the procedure is feasible and useful as a rough guide in determining Dicumarol dosage If further studies confirm the validity of this method anticoagulant therapy need no longer be denied a patient because of expense inconvenience or lack of laboratory facilities

Because of the disadvantages of heparin and particularly of Dicumarol several new anticoagulant drugs have been introduced and are receiving a clinical trial *Paritol*[238] a polysulfuric acid ester of polyanhydro mannuronic acid is used intravenously and has been shown to prolong the clotting time for from eight to twelve hours Its anticoagulant effect persists 2 to 3 times as long as does that of heparin and it is only necessary to give one injection every eight to twelve hours The dosage is about 7 times that of heparin It should not be administered to patients with renal disease and there have been occasional mild reactions to the drug

Tromexan[39 41 111] *an ethyl ether of Dicumarol acts much* more quickly than does Dicumarol and is not cumulative Its effect is apparent in eighteen to twenty four hours and when used with heparin the latter drug may be stopped after the first day The dose used is about 6 times that of Dicumarol *We have found it difficult to maintain adequate* prolongation of the prothrombin time with tromexan but experience with this drug is not yet sufficient for a complete evaluation of its usefulness

The careful use of anticoagulant therapy has greatly decreased the incidence of embolization in our patients

We have also decreased the incidence of embolization by permitting patients with myocardial infarction to move about more freely While in bed the patients are advised to move their legs frequently if using a bed pan is difficult they are permitted to use a bedside commode after the second day if their condition is satisfactory They are cautioned never to bear down or strain and to prevent distention laxatives (mineral oil cascara or Milk of Magnesia) are given freely after the second day following the occlusion If a bowel movement does not occur after three days a low enema is given In some instances it may be necessary to administer laxatives as well as enemas every two to three days during the first two weeks of bed rest

If after three weeks the electrocardiogram has stabilized

the white count has become normal the patient is afebrile and no complications have occurred the patient is graduallv permitted to walk We do not depend upon the sedimentation rate to determine the total period of bed rest since frequently it does not return to normal for two or three or even twelve months *i e* long after the patients are ambulatory and have returned to work We feel that early ambulation has also greatly contributed toward lowering the incidence of embolization

If embolization occurs despite these measures the anticoagulants are continued In cases of cerebral embolization caution must be observed in administering these drugs If a spinal tap does not reveal hemorrhagic fluid and if it is feared that further emboli will occur further anticoagulant therapy is justified Sedation should be avoided in these patients and caffeine sodium benzoate $7\frac{1}{2}$ gr (0 5 gm) intramuscularly every 4 hours and aminophyllin 3 gr (0 2 gm) intramuscularly every 4 hours may be given Other supportive measures should be employed oxygen should be used and parenteral feedings continued until the patient is able to feed himself

When a peripheral embolism occurs intra arterial papaverine 50 to 100 mg every two to four hours given proximal to the occlusion is sometimes of value in increasing circulation in the affected extremity We have recently obtained excellent results with intra arterial Priscoline 50 mg (2 cc) every four to six hours Various ganglionic blocking agents also produce a high degree of vasodilatation and reduce vasospasm when administered intravenously We have found that the Methonium derivatives C 5 (bis-trimethyl ammonium pentane dibromide) and C 6 (bis trimethyl ammonium hexane dibromide) produce a longer lasting blockade and are less toxic than Etamon (tetra ethyl ammonium chloride) C 5 (10 mg cc) and C 6 (25 mg cc) mav be administered in 2 3 cc doses intravenously every 4 hours to maintain an adequate blockade If these drugs are not available Ltamon (100 300 mg) mav be given every 4 hours or repeated sympathetic nerve blocks

employed Although we have not as yet used intravenous Dibenamine in this condition this drug should be of great value as its adrenergic blocking effect usually lasts from 36 to 72 hours after a single dose of 5 to 7 mg /kg In view of this fact Dibenamine will probably prove to be superior to other medications presently in use If the patient is able to tolerate oral therapy the measures outlined above may be supplemented by the use of Priscoline 50 to 75 mg every 4 hours 688A (N N Dibenzyl betachlorethylamine hydro chloride) a new Dibenamine derivative which is an orally effective sympathetic blocking agent [97] has not as yet been tried clinically in this condition but this should be done in the future If the patient is not seen early and if gan grene has already occurred the leg should be packed in ice in preparation for amputation

Embolectomy is the treatment of choice if it can be performed within eight to twelve hours after the acute epi sode In expert hands results have been good and even massive saddle emboli lodged at the bifurcation of the aorta have been removed successfully [39] It is a serious operative procedure particularly in patients with recent myocardial infarction but it is justified unless the patient is critically ill A patient recently observed illustrates the fact that embolectomy can be performed successfully even in the presence of serious heart disease

A sixty three year old tailor developed an acute coronary occlusion with anterior infarction He did not receive Dicumarol On the fifth day he complained of severe pain in the right leg which became mottled and cold up to the mid thigh The femoral pulse was not palpable Five hours after the onset of symptoms operation was performed by Dr Gabriel Seley of New York and an embolus was re moved from the femoral artery Complete function of the extremity was restored and the patient made an uneventful recovery

If embolectomy is not feasible and amputation becomes necessary it should be performed under local or block anes thesia after demarcation has occurred

Chapter

4

SYNCOPE

SYNCOPE occurs in many conditions, and usually in patients without organic heart disease (Table IV) Two main mechanisms account for the temporary cerebral anoxia, which is the physiological basis of syncope (1) a sudden fall in cardiac output, e g following severe hemorrhage [36] (2) a marked decrease in peripheral resistance and a fall in blood pressure with a fairly well maintained cardiac output, e g, neurogenic fainting [158] Neurogenic or vasodepressor syncope, by far the most common is produced by reflex peripheral vasodilatation, and may be precipitated by the sight of blood or by fright, pain or severe emotional disturbances [70] It is more frequent in men than in women Premonitory symptoms such as nausea sweating or lightheadedness usually precede the attack which occurs only if the patient is standing This type of syncope can usually be terminated by laying the patient down with his head lowered If the syncope lasts for more than ten to fifteen seconds clonic twitching may appear but true grand mal seizures are extremely rare In this type of attack it is sometimes necessary to administer sympathomimetic drugs, i e, epinephrine, 0 5 cc of 1 1000 solution subcutaneously

Another form of syncope due to peripheral vasodilatation is observed in patients who while sitting or recumbent employ heating pads for low back pain When these patients suddenly resume the erect position a temporary cerebral anoxia occ[illegible] and fainting may result Fainting

TABLE IV.—DIFFERENTIAL DIAGNOSIS AND TREATMENT OF SYNCOPE

Type	Precipitating Factor	Premonitory Symptoms	Duration of Attack Convulsions†	Laboratory Findings	Treatment
1) Vasodepressor syncope a) Neurogenic	Fright, pain, emotional upset, sight of blood	Nausea, sweating, lightheadedness, dizziness (only occurs when patient is standing), premonitory phase 1-5 minutes	Usually of short duration 1-15 seconds—after 15 seconds twitching may appear	Cardiac output normal Blood pressure falls Pulse rate may rise or fall Electroencephalogram abnormal	Usually terminated by laying patient down with head lower than feet Rarely .5 to 1 cc. Epinephrin (subcutaneously) if necessary
b) Carotid Sinus Syncope	a) Pressure on neck b) Turning head c) Cerebral Arteriosclerosis d) Digitalis	*Usually no premonitory symptoms*	Short duration Convulsions may occur	Blood pressure falls Pulse rate decreases Electroencephalogram abnormal	Ephedrine Sulfate 25 mg. t.i.d. or Atropine .3 mg. t.i.d. Excision of sinus nerves if attacks are severe and persist
c) Secondary to Other Factors	Internal or external hemorrhage, fevers	Same as (a)	Same as (a)	Same as (a)	Treat primary condition
2) Postural Hypotension	a) Tabes Dorsalis b) Spinal Cord Tumors c) Addison's Disease d) Diabetes Mellitus e) Standing in one place, arising suddenly f) Idiopathic	Lightheadedness, dizziness	Usually of short duration (1-5 seconds) No convulsions	Blood pressure falls Pulse rate increases Electroencephalogram abnormal	Lay the patient down Tight abdominal belt and neosynephrine or ephedrine Tilt bed Caution against rising quickly Treat primary disease
3) Hysterical Syncope	a) Suggestion b) pressure on any area of body c) tense situation d) Hyperventilation	*No premonitory symptoms*	*Never any convulsions* *May last for hours*	No change in blood pressure or pulse rate No electroencephalogram changes	Psychiatric
4) Cardiac Syncope	a) Heart block with periods of asystole b) Ventricular tachycardia or other arrhythmias	Dizziness, palpitations, chest pain, nausea, epigastric pain	Usually short (5-10 seconds) May last longer with convulsions	Electrocardiographic abnormalities Blood pressure falls Pulse rate may be extremely slow, rapid or imperceptible	Treat cardiac arrhythmia: Quinidine, Digitalis, Pronestyl For heart block: Epinephrin, Atropine, etc. See text
Hypoglycemic Syncope	a) Islet cell tumor b) Overactive pancreas c) Too great an interval between meals d) Hepatic disease	Weakness, extreme hunger, tiredness, sweating, pallor	May be prolonged if not treated Convulsions occur frequently	Blood sugar extremely low Electroencephalogram abnormalities Pulse rate increased Blood pressure unchanged or low	Intravenous glucose, then oral glucose—depending upon etiology (see text) a) remove tumor if present b) low carbohydrate diet c) high carbohydrate diet if liver disease is present
Tussive	a) Severe coughing b) Bearing down	Dizziness, tightness	Short (1-5 seconds)	No unusual findings	Warn patient against "bearing down" or excessive coughing [illegible]

occasionally occurs in normal persons who are required to stand in one place for long periods of time and in patients with autonomic nervous system disturbances *i e* over active vagal reflexes (carotid sinus syncope) or poor sympathetic nervous regulation of blood vessels[34 244] (postural hypotension)

Many persons with a hypersensitive carotid sinus faint when the sinus is massaged[243] but only a small percentage of these experience spontaneous syncope as a result of this sensitivity Consequently a diagnosis of carotid sinus syncope should be made only if the following criteria are satisfied (1) The attacks must occur without the premonitory signs usually seen in the type of vasodepressor syncope not associated with carotid sinus hypersensitivity (*i e* syncope due to fright or sight of blood) (2) The attacks should be reproducible by sinus stimulation (3) The attacks should not occur after the administration of adequate doses of atropine or ephedrine *i e* doses large enough to prevent bradycardia on manual carotid sinus stimulation True cases of this syndrome are relatively uncommon when compared with other types of vasodepressor syncope

An interesting illustration of carotid sinus sensitivity is furnished by a seventy one year old man who was admitted to the hospital because of four attacks of syncope during the preceding four months Examination revealed a blowing systolic murmur at the aortic area transmitted into the neck The blood pressure was 160 80 The peripheral pulses were not palpable The electrocardiogram showed evidence of previous lateral wall infarction In the hospital he experienced brief episodes of dizziness It was thought that his syncope was the result of cerebral arteriosclerosis and aortic stenosis *Pressure on the right carotid sinus however* produced asystole and a drop in blood pressure to 80 60 The patient became blanched his hands twitchd and he yawned Stimulation of the left carotid sinus produced similar results but with less regularity He was given oral

atropine 3 mg (gr 1/100) q i d and after several days carotid sinus pressure caused no change in the heart rate (Fig 12) The patient has been maintained satisfactorily on this medication It was extremely difficult to establish a diagnosis of *true carotid sinus syncope* in this case despite the fact that definite *hypersensitivity* of the sinus could be demonstrated The patient had cerebral arteriosclerosis and aortic stenosis which alone may have caused the syncope

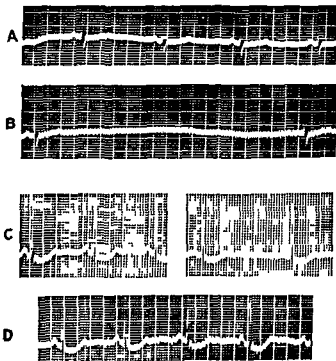


FIG 12 —J Q m 61 Arteriosclerot c heart d sease Cont nuo s lead II 1 Control s n s bradycard a rate 52 *B* After pre re

t on

Since atropine definitely prevented the attacks however, the diagnosis of syncope secondary to carotid sinus sensitivity, was the most logical one Patients who have extensive arteriosclerosis and are receiving digitalis are more susceptible to syncopal attacks and, in some instances stopping the digitalis will eliminate the attacks completely Carotid sinus syncope may be induced by pressure on the sinus from any cause *e g* by an enlarged gland in the neck or by a sudden movement of the head

Tight collars often serve as a trigger mechanism J C, a thirty eight year old midwestern preacher was investigated because he had fainted several times while delivering his Sunday sermon In eliciting his history it was discovered that during the week he worked in the field with his collar open on Sunday, however he wore a stiff collar to appear in church Carotid sinus pressure repeatedly induced asystole and syncope in this patient and the attacks were eliminated when the patient stopped wearing tight collars

Convulsions may occur as a result of excessive carotid sinus stimulation [264] and caution must be observed when testing a patient for sensitivity especially an older person or one with arteriosclerosis Pressure should never be applied for more than ten seconds and the patient should be recumbent when tested Ephedrine sulphate (25 mg) Paredrine (60 mg 3 times a day) or atropin sulphate (0 3 mg grain 1/200), will prevent most attacks Neosynephrine, in oral doses of 20 to 25 mg 3 times a day is occasionally successful when other medication fails When it is necessary to use sympathomimetic drugs *i e*, ephedrine for long periods of time, it is wise to administer small doses of sedatives (barbiturates) simultaneously to prevent nervousness and insomnia In a few cases excision of the sinus has given permanent relief [150]

Postural hypotension may occur in normal persons, in patients with neurocirculatory asthenia and in those with serious underlying central nervous system disease,[68] such as cord tumor or tabes dorsalis [237] We have also observed

extremely severe postural hypotension in patients with advanced diabetes mellitus Addison s disease and pheochromocytoma When these people assume the upright position the normal compensatory splanchnic constriction does not occur the cardiac output is diminished and the blood pressure falls

In patients with chronic postural hypotension and recurrent syncopal episodes therapy is extremely difficult The primary disease should be sought and treated if possible *i e* Addison s disease varicose veins *etc* If no primary condition is found atropine epinephrine neosynephrin or Paredrine should be tried in the doses cited These drugs have not proven very successful however and are usually more effective when used in conjunction with a tight abdominal belt and or elastic bandages around the lower extremities It is occasionally temporarily helpful for these patients to sleep in a bed tilted 20 to 25 degrees from the horizontal with the head up [64] Desoxycorticosterone acetate and sufficient salt to cause slight dependent edema sometimes reduce the frequency of attacks The patients should be instructed to rise from bed slowly and to avoid taking drugs that cause peripheral vasodilation *i e* amyl nitrite or nitroglycerin

Fainting may be hysterical in origin particularly in women This type of syncope is not accompanied by premonitory symptoms and is not associated with any changes in pulse or blood pressure These patients may fall to the ground and remain unconscious for a period of several seconds or hours but convulsions do not occur and injury rarely results Electroencephalographic changes which are seen in most other forms of syncope that last more than a few seconds do not appear in this condition [70] Fainting may be precipitated by suggestion or by manipulation of almost any part of the body Therapy is mainly psychiatric

In anxiety states syncope occasionally occurs as a result of hyperventilation Such an attack may be reproduced by having the patient breathe deeply for one or two minutes

Assurance and sedation are the only treatments of value in correcting this condition

A rare form of syncope is that which follows severe bouts of coughing or the Valsalva maneuver (a forced expiration with the glottis closed — the bearing down effort)[153] A marked rise in intrathoracic pressure occurs and prevents adequate filling of the right atrium resulting in a marked fall in cardiac output and cerebral anoxia Recognition of this type of syncope is important since most of these attacks may be prevented by warning the patient against excessive bearing down efforts such as straining at stool and against remaining upright during a spell of severe coughing

Fainting which occurs early in the morning before breakfast or several hours after a meal may be the result of hypoglycemia This type of syncope is seen in cases of hyperinsulinism caused by pancreatic adenomata in patients on a high carbohydrate diet with an overactive pancreas and in cases of marked hepatic disease[45] The patient usually experiences weakness dizziness and extreme hunger before fainting convulsions often occur if therapy is not instituted quickly Intravenous or oral glucose should be given as soon as possible and recovery from the attack is usually rapid The diagnosis is made from the history the fasting blood sugar determination and the glucose tolerance test Extremely low blood sugar levels may be obtained during the course of the latter test, especially in the two three and four hour specimens and fainting may occur before the test can be completed These attacks may be prevented in several ways depending upon the etiology of the condition If hypoglycemic attacks occur before breakfast and rarely during the day and if liver damage is demonstrated a high carbohydrate high protein diet should be prescribed If the attacks occur before breakfast and frequently during the day and if they become progressively more severe and are not relieved when the patient is put on a low carbohydrate high protein diet then a pancreatic adenoma is probably present and surgical removal of the tumor is advisable

If the attacks occur approximately two to three hours after meals, and if no evidence of liver disease or progression is found, then so called "functional" hypoglycemia is present, and the patient should be put on a low carbohydrate, high protein diet with frequent feedings Additional points of differentiation between these three major types of hypo glycemia, and suggestions for therapy may be found in a recent review [46]

In organic heart disease syncope occurs in complete heart block, *i e*, Stokes Adams attacks, and sometimes at the onset of paroxysmal tachycardia[47] (Chapter 1 p 11) and following an acute coronary occlusion In patients with calcific aortic stenosis secondary to rheumatic or arterio sclerotic heart disease fainting may also occur especially following exertion The prognosis of syncope in organic heart disease is, of course, more serious than it is in non cardiac cases Therapy in the former group is directed against the primary condition

Chapter

5

RHEUMATIC HEART DISEASE

During the acute stage of rheumatic fever the occurrence of acute pericarditis congestive failure and arrhythmias frequently necessitate emergency treatment In such cases early adequate treatment often reduces the morbidity and mortality rate Previously only salicylate therapy and symptomatic treatment were given The recent introduction of ACTH and Cortisone however has provided the physician with drugs that appear to be extremely useful and rapidly effective in the treatment of the acute rheumatic process[2, 3, 70, 269] For this reason these drugs should be used whenever possible in cases of active rheumatic carditis that are seen early

We have recently observed a critically ill patient in severe left and right heart failure with rheumatic pancarditis who did not respond to the usual therapy but who improved remarkably after receiving 25 mg of ACTH every six hours for six days There was little doubt that death would otherwise have occurred After six days the daily dose of ACTH was *reduced from 100 to 60 mg and this was maintained* for seven days The daily dose was then further reduced to 40 mg This treatment was continued for two more weeks and the patient recovered

A course of treatment of from seven to ten days has been suggested to control the carditis[69] but we feel that ACTH should be continued for a period of at least two to three weeks as was done in the case cited If this does not prove effective another course of therapy may be given If

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alter the subsequent course of rheumatic heart disease *i e* the development of valvular lesions congestive heart failure *etc* is questionable

Should *congestive failure or arrhythmias* occur during the course of acute rheumatic fever they are treated as has already been outlined Digitalis however should be used *with caution in these cases This drug is often ineffective* in the acute phase of rheumatic carditis and may even be *harmful furthermore the tendency is to push the dosage* to toxic levels in order to obtain an effect [8] It is therefore *wiser to depend upon salt restriction and mercurial diuretics* in congestive failure before digitalization is attempted If *failure persists however despite these measures digitalis* may be used Although some observers[2] have reported *favorable results with digitalis in the presence of active* carditis we are unable to confirm these observations The *drug is occasionally effective in cases with congestive heart* failure and auricular fibrillation but its administration *should be carefully controlled*

Acute pericarditis may be associated with severe precor*dial or epigastric pain which is relieved only by morphine* The pain usually develops within a few days after the onset *of acute rheumatic fever The heart sounds are distant a* friction rub may be heard and shock may occur The *electrocardiogram usually presents a typical pattern con*sisting of RS T elevation in two or more leads without *Q waves (Fig 13) If an effusion accumulates the rub* may disappear and the patient become more dyspneic and *orthopneic Tapping is rarely necessary in these cases* however and has been performed only when a rapidly *progressive increase in dyspnea and venous pressure occurs* as a result of cardiac tamponade Usually the fluid is *resorbed spontaneously as the acute infection subsides* on bed rest and drug therapy

In the course of chronic rheumatic heart disease with mitral stenosis and auricular fibrillation auricular thrombi *may form and in rare instances occlude the valve open*

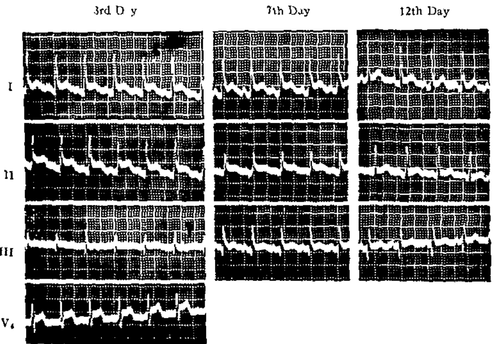


FIG 13 —M P f 26 Acute rheumatic fever with pericarditis RS T elevations are present in all leads

ing[77, 171, 276] (ball valve thrombus), producing a serious emergency. Cyanosis appears at the tip of the nose, the lobes of the ears, and the fingers and toes and severe dyspnea, congestive heart failure, and shock usually develop. Death may occur suddenly[6] or after a few hours or days. The treatment is supportive, including oxygen and the usual measures employed in congestive failure and shock. Morphine 15 mg ($\frac{1}{4}$ gr), may relieve the restlessness. Occasionally a change in position brings relief to the patient. We have seen the cyanosis disappear immediately when the patient sat up and leaned forward.

Pulmonary infarction and cerebral and peripheral arterial occlusion may also be caused by auricular thrombi that are released into the blood stream. One embolic episode is often followed by others. In such cases, chronic Dicumarolization is indicated. The dosage is adjusted while the patient is hospitalized, for the next month prothrombin times are done 3 times a week, thereafter, weekly determinations are sufficient. The results reported with this treatment appear to be satisfactory.[106, 216] We have observed three patients during the past eighteen months who had experienced repeated bouts of embolization, and who had consequently been continuously bedridden. They were hospitalized and treated with Dicumarol being maintained on the drug after discharge. They have now been followed for from nine to eleven months, and have not experienced a single embolic episode, a remarkable result in view of the previous course of their disease. Despite the fact that chronic Dicumarolization may markedly decrease the number of embolic episodes this procedure should not be carried out if adequate facilities for determining prothrombin times are not available. Chronic anticoagulant therapy with the drugs now in use is by no means a simple, safe procedure, and should be embarked upon with great caution.

In 1946, Dock[67] suggested left auricular appendectomy or ligation in patients with mitral stenosis in order to reduce

the high incidence of embolization since he believed that most thrombi originated in this appendage Recently the auricular appendage has been tied off successfully in five cases [12,160] although the results were poor in the first three [16] In view of the fact that thrombi may form in both appendages in cases of rheumatic heart disease [133] especially in those with congestive heart failure it is quite possible that bilateral auricular resection will prove to be the correct surgical procedure to prevent embolization Since neither *of these operations is a simple one follow up studies will be* necessary to justify their use The results thus far obtained suggest that the continual use of anticoagulants produces the same effect as auricular resection without its serious drawbacks for this reason we favor the former treatment in these cases

In rheumatic heart disease embolization should suggest the presence of subacute bacterial endocarditis The emboli are generally small and occlude small peripheral arterioles producing white centered petechiae Every patient with rheumatic heart disease who runs an unexplained fever *for more than one week should have blood cultures taken* Three blood cultures may be taken during the first day it is unnecessary to wait twenty four hours between cultures as is commonly done If a patient with rheumatic heart disease exhibits white centered petechiae or splenomegaly in addition to the fever a diagnosis of subacute bacterial endocarditis is justified even if blood cultures are negative and therapy should be promptly instituted [89] In a case recently observed by us cerebrovascular embolization occurred because treatment was delayed pending the report of the blood cultures In any case with clinical findings of subacute bacterial endocarditis therapy should be started after two blood cultures have been taken *i e* eight hours after hospitalization

Recently in patients with tight mitral stenosis several surgical procedures *have been employed in an effort to* decrease the incidence of intractable congestive failure

hemoptysis and recurrent attacks of pulmonary edema. Such attacks are extremely frequent in patients with mitral valvular lesions, especially following activity.[10] An extracardiac shunt to prevent pulmonary edema by means of an anastomosis between a branch of the inferior pulmonary vein and the azygos vein has been performed with some good results,[27] but this operation has now been replaced by direct operation on the mitral valve.

Successful operative repair of the mitral valve has been done[110] in patients with mitral stenosis whose cardiac output was low and whose pulmonary venous pressure was markedly elevated, i.e., patients with a "tight" mitral stenosis with little insufficiency. In cases with normal cardiac output, i.e., with a lesser degree of stenosis, it is sometimes better to create an interatrial septal defect. When patients are too sick to be operated on, repeated sympathetic blocks may be of value in preventing paroxysmal tachycardia and/or acute pulmonary edema.[110] Much greater experience is required before definite conclusions can be drawn regarding the operative correction of mitral stenosis.

Chapter

6

HYPERTENSIVE ENCEPHALOPATHY AND CRISES

HYPERTENSIVE crises may occur in the course of essential hypertension, particularly in its malignant phase, and in acute nephritis. The patient becomes acutely ill with unremitting headache and may exhibit temporary blindness, paralysis, aphasia and convulsions. These attacks are often brief but if they persist relief may occasionally be obtained by the intravenous administration of 20 cc of a 10 per cent solution (2 to 4 gms) of magnesium sulphate, by withdrawal of spinal fluid or by the intravenous injection of 50 cc of 50 per cent dextrose. Good results have also been achieved with intravenous Dibenamine (5 to 7 mg per Kg in 100 cc of 5 per cent glucose in water given in one hour).[191, 575] This drug lowers the blood pressure; one dose may alleviate the mental symptoms for several days. The infusion of Dibenamine may be repeated every forty-eight to seventy-two hours to keep the patient symptom free.

Pheochromocytoma, a tumor which secretes both epinephrine and nor-epinephrine, is usually found in the adrenal medulla or the retroperitoneal chromaffin tissue. It may cause attacks of severe hypertension with headaches, palpitations, tachycardia, vertigo, abdominal pain, nausea, vomiting and profuse sweating.[19] Because of the increased amount of circulating adrenalin in patients with a pheochromocytoma an elevation of the basal metabolic rate sometimes occurs. Occasionally glycosuria and an elevated blood sugar are present as a result of an increased glycogen

breakdown secondary to epinephrine action The attacks of palpitation sweating *etc* may be sudden in onset and occasionally can be induced by palpating the region of the kidneys The patient becomes acutely ill and pale and breaks out in a cold sweat In cases of pheochromo cytoma *the hypertension may be persistent and chronic headaches and continuous sweating may be present* The diagnosis of this tumor should be considered in hyper tensive patients who perspire profusely or who have an elevated basal metabolic rate a glycosuria or a marked loss of weight It may be confirmed by a positive response to histamine[illegible] or tetra-ethyl ammonium chloride (Eta mon)[148] *i e* a rise in blood pressure and a reproduction of the acute attack The rise in blood pressure is probably the result of a dilatation of blood vessels in the tumor and an increased outpouring of adrenalin Benzodioxane[93] and Dibenamine[1 9] which cause a fall in blood pressure and bring relief of symptoms as a result of their adrenolytic action are also extensively used as aids in establishing the diagnosis Benzodioxane at present appears to be the most satis factory diagnostic agent during the hypertensive stage of the disease *A false positive reaction rarely occurs when it is* used If hypertension is not present when the patient is tested Etamon is preferable to histamine because it pro duces a less violent rise in blood pressure Although calci fication in this tumor is uncommon its presence in the region of the adrenal gland may suggest the true diagnosis in a hypertensive patient with the above mentioned symp toms[197] Since hypertension secondary to a pheochromo cytoma is the only type of hypertension that can be cured by surgical intervention we feel that *all* patients with either *fixed or paroxysmal hypertension should be studied for the* presence of this tumor The testing agents now available are simple to use and do not necessitate hospitalization of the patient

The acute attacks should be treated with intravenous Dibenamine given as described above The tumor should be excised as soon as possible

Chapter

7

DISSECTING ANEURYSM

DISSECTING aneurysm of the aorta is relatively uncommon but its diagnosis is important for two reasons (1) if an erroneous diagnosis of acute coronary occlusion or pulmonary infarction is made and anticoagulant therapy is given the possibility of recovery from a dissecting aneurysm is further reduced (2) if a method for the surgical treatment of dissecting aneurysms is perfected early diagnosis will be essential for success Only a small percentage of the cases is diagnosed antemortem probably because of the failure to suspect the presence of the condition Contrary to general belief a dissecting aneurysm is not invariably fatal 10 to 25 per cent of patients have lived for from three months to eight years following its onset [55] The dissection may be acute lasting only a few hours and terminate in death as a result of rupture of the aorta or it may be subacute causing repeated bouts of pain for a period of 2 to 3 days and end in recovery

The attack does not appear to be precipitated by unusual exertion usually occurs in patients with hypertension and is sudden in onset Occlusion of a branch of the aorta *i e* iliac intercostal or coronary is common There is a sharp knife like pain in the chest and/or back with occasional radiation to the epigastrium the lower abdomen the flanks and the legs Syncope is noted in 10 per cent of the cases *and shock may occur quickly The patient may* writhe in pain but unlike the pain of coronary occlusion this pain is not crushing or constrictive and does not

require a minute or two to reach its maximum intensity Various neurologic signs may appear including paraplegia which is caused by hemorrhagic infarction of the spinal cord [230] An aortic diastolic murmur is heard in many cases Electrocardiographic evidence of left heart strain or coronary insufficiency may appear or of coronary occlusion if the dissection occludes a coronary artery A coronary occlusion secondary to a dissecting aneurysm may sometimes be differentiated from the ordinary type of coronary occlusion by the presence of pain in the back the lower abdomen or the legs by the sudden appearance of a diastolic murmur or by the presence of neurologic changes If a pleural or pericardial effusion or hemorrhage into the wall of the aortic arch occurs a widened mediastinum may be detected on fluoroscopy or *x* ray This procedure should be carried out with great caution and actually should not be used if the diagnosis is fairly well established from other findings The diagnosis should be considered in any patient with hypertension who suddenly develops severe knife like pain in the chest back epigastric region or legs and in whom a diastolic murmur suddenly appears

A case in which the correct diagnosis was suggested during life was that of a hypertensive man of 42 who developed episodes of severe substernal pain and shock with apparent complete recovery between attacks The electrocardiogram showed the deep Q waves and RS T elevations characteristic of coronary occlusion On the third day during one of his attacks the heart sounds suddenly became faint and the area of cardiac dullness increased A pericardial tap revealed hemopericardium and a diagnosis of dissecting aneurysm was made He died almost immediately and postmortem examination disclosed a dissection involving a coronary artery

Many times however the history and findings are completely atypical and a correct diagnosis cannot be made For example A sixty three year old white male complained of sudden severe intermittent pain in the left lower

quadrant He had experienced mild pain in that region eight months before for a period of four or five days but the pain had not recurred until the present admission Examination revealed an ill defined mass in the left lower quadrant His blood pressure was 170 100 and no murmurs were heard A diagnosis of carcinoma of the colon or diverticulitis was made Several days later the patient was found to be quite pale and the mass appeared to be larger Twenty four hours later he died suddenly Necropsy showed evidence of dissection of the aorta from the thoracic to the abdominal portion with rupture of the abdominal aorta and retroperitoneal hemorrhage

When the presence of a dissecting aneurysm is suspected the patient should be put to bed immediately Repeated doses of morphine should be given if necessary to attain complete sedation He should be kept in bed for at least three weeks and then should gradually be permitted to walk Anticoagulants should not be used If an accurate diagnosis is made and if treatment is instituted early a much larger percentage of cures may be effected

In view of the tremendous advances in vascular surgery it is not unlikely that a procedure will be developed to re channel the blood in the dissected coats of the aorta into the lumen of the vessel A prerequisite to this however is the discovery of methods to detect the site and path of the dissection

Chapter

8

TRAUMATIC HEART DISEASE

The heart may be injured directly by penetrating bullet or knife wounds or indirectly by severe blows to the chest [10 17]

The diagnosis of a **penetrating wound of the heart** is simple if external evidence of the injury is present Occasionally, however, a bullet or a needle which had entered the body at some remote point, may be carried to the heart through the veins,[101] and produce cardiac damage In these cases the diagnosis is often difficult If a patient with a recent penetrating injury of the chest wall presents evidence of blood loss congestive heart failure or shock trauma of the heart should be suspected The commonest result of such trauma is hemorrhage into the pericardial sac or laceration of the myocardium with infarction In rare instances valvular rupture may occur When a hemopericardium develops, fluoroscopy or x ray may show evidence of a widened cardiac shadow with diminished pulsations or electrocardiograms alone may show characteristic changes Occasionally findings in such cases are similar to those seen in myocardial infarction Surgical intervention is justified only when there is evidence of increasing hemopericardium and cardiac tamponade *i e*, shock, with a weak thready pulse and drop in blood pressure (low cardiac output), or congestive heart failure A pericardial tap should be performed immediately, and as much blood as possible should be aspirated [214] If blood reaccumulates, or if the symptoms

persist or recur, a severe myocardial laceration has occurred and should be repaired immediately.

Shock should be treated as efficiently as possible before and during the operation. The Trendelenburg position, frequent transfusions of blood (plasma if blood is not available), and the liberal use of morphine intramuscularly or intravenously will often help the patient to surmount the crisis until the wound is repaired. When plasma or blood is not immediately available, intravenous infusions of 10 per cent glucose in water may be given in the meantime. Despite the presence of cardiac tamponade, the cardiac output may be increased and the patient's condition improved by these measures.[9]

Excellent reviews of the technique of repairing lacerations or removing foreign bodies have been written.[7, 9] These surgical procedures need not be discussed here. Complications such as cardiac arrest encountered during operation are discussed in Chapter 9. In experienced hands the operative mortality rate in such cases is 25 to 30 per cent.[6] The site repaired heals and a firm scar develops. Despite the fact that a major coronary artery is often ligated, infarction does not always occur and cardiac function is usually good.[17] A case seen by us demonstrated this finding. W. C., a thirty-five-year-old laborer, was stabbed in the region of the heart with a long knife two hours prior to admission. There was profuse hemorrhage but the patient walked several blocks to the hospital. He complained of pain in the chest but was neither dyspneic nor cyanotic. The blood pressure was 100/80. The heart sounds were distant and a pericardial rub was heard which soon disappeared. An *x*-ray of the chest and an electrocardiogram were negative. In view of the profuse bleeding an exploratory operation was performed. A laceration of the parietal pericardium with a moderate hemopericardium and an insignificant stab wound of the anterior surface of the heart were found. Upon gentle manipulation the oc

cluding blood clot was dislodged and free bleeding occurred this could be controlled only by the insertion of three sutures, two of which entered the chamber Following the operation, *the electrocardiogram showed RS–T elevation* followed by T wave inversion in the standard leads (chest leads were not taken because of the chest strapping) the typical serial changes of acute pericarditis In addition, a Q wave appeared in lead II this produced a W shaped QRS which was still present a year later when the T waves in the standard leads had returned to normal It is possible that the alteration in the QRS was produced by the injury associated with the suturing Despite the fact that a coronary artery had been ligated during the operation the patient made an excellent functional recovery

Nonpenetrating chest injuries may produce signs and symptoms that are difficult to distinguish from those found in spontaneous coronary occlusion or coronary insufficiency The causal relationship between trauma and myocardial damage is frequently difficult to establish As a rule only a very severe blow to the chest wall produces a detectable cardiac injury Many boxers and victims of accidents who sustain injuries to the chest show no anatomical or electrocardiographic evidence of myocardial damage [44, 100]

Many cases of alleged traumatic coronary occlusion reported in the literature probably represent instances of the coincidental occurrence of coronary occlusion and trauma *The electrocardiograms which had been reported* in a large number of these patients as showing an occlusion actually showed only ST and T wave changes which occur in coronary insufficiency and not in occlusion [208 p 8] In some *instances a careful history will elicit the fact that the pre*cordial pain preceded the accident Although we are certain that episodes of coronary insufficiency may be precipitated by effort or trauma we believe that the occurrence of coronary occlusion is *coincidental with extreme physical* exertion and that the occlusion had occurred prior to the injury [17, 173] Trauma to the chest wall occasionally may

produce subendocardial tears and myocardial contusions in otherwise normal hearts[120] All cases of so called steer ing wheel accidents should be carefully observed for several days in a search for electrocardiographic or clinical evi dence of heart damage even in the absence of rib fracture or severe external injury[47] These patients may show var ious arrythmias without other changes for several days following the accident All cases with a definite history of severe chest trauma that develop precordial pain or dis comfort at the time of the injury should be suspected of having sustained a myocardial contusion the patients should therefore be put to bed and be given adequate sedation

The following criteria help one in determining whether the cardiac damage was caused by a specific trauma[147] The decision must be made on the basis of the facts in each case but in general we require the presence of two or more of these criteria for causal relationship

1 Significant electrocardiographic changes must be present
2 Cardiac enlargement unexplained by pre existent cardiac disease or hypertension must appear
3 Abnormal rhythms not including premature beats unless they are accompanied by other electrocardio graphic changes must occur
4 A pericardial friction rub must be heard or evidence of effusion found
5 Congestive heart failure precipitated by the blow or strain must develop
6 Clinical signs and laboratory evidence of coronary insufficiency and or myocardial infarction must be come manifest within twenty four hours following the incident

Such patients should be treated in the same manner as patients with nontraumatic coronary insufficiency or occlu sion Anticoagulant drugs should be withheld however

because of the frequent occurrence of pericardial hemorrhage If hemopericardium does occur the blood should be repeatedly aspirated if necessary

Rupture of the heart occurs in a few of these cases within ten days to two weeks after the trauma but if the patient is carefully treated and put to bed as soon as symptoms appear, the incidence of this fatal complication may be reduced

Many cases of traumatic myocardial damage recover completely after three to five weeks of bed rest, and show no clinical or electrocardiographic evidence of heart disease for years after the accident

Chapter

9

SURGICAL EMERGENCIES IN CARDIO VASCULAR DISEASE THEIR PREVENTION AND TREATMENT

Surgery in the Cardiac Patient —The need for surgical treatment of an intercurrent disease among cardiac patients often arises This need has increased in frequency with the increase in the number of the aged many of whom suffer from some cardio vascular disorder Careful medical supervision of such patients is necessary in order to prevent the development of acute congestive failure coronary insufficiency tachycardias and pulmonary embolism following operation

Most of the patients who have heart disease tolerate an operation well the exceptions being those who are in intractable failure or are recovering from a recent myocardial infarction Serious complications do not develop even in these patients if the pre and postoperative treatment is properly administered

A seventy eight year-old man who was studied because of increasing difficulty in urination exemplifies this point A firm enlarged prostate was found and a prostatectomy was suggested but the patient was in severe congestive heart failure He was rapidly digitalized and placed on rigid salt restriction A complete A V block with a ventricular rate of 36 developed and the patient experienced several Stokes Adams seizures until the digitalis was stopped *The danger involved in the operation was ex*

plained to the patient, but he insisted on its being performed During the operation profuse hemorrhage occurred and the patient went into mild shock A continuous transfusion was administered together with repeated small doses of ephedrine and neosynephrine intramuscularly The patient received 2500 cc of blood within twenty four hours without developing congestive failure There were no further Stokes Adams seizures He made an uneventful recovery, and has remained well during the past four years

Age should not be a deterring factor in patients who require surgery[204] Many elderly patients with heart disease and resultant severe cardiac disability, moreover are improved by the surgical removal of foci of infection and irritation M S, a fifty nine year-old housewife who had been an invalid because of severe recurrent attacks of angina pectoris, also suffered from biliary colic The surgeons were hesitant about operating on her because of her cardiac condition but they were finally prevailed upon to do so She tolerated the operation very well and was then practically free of her anginal attacks for two years M R a sixty year old woman with gallbladder disease suffered an acute posterior wall infarction Her convalescence was marked by repeated attacks of biliary colic A cholecystectomy was performed one month after the occlusion had occurred Her postoperative course was uneventful and since then she has not complained of pain

In the presence of severe heart failure or after a recent coronary occlusion an operation should be performed only when it is urgently needed, and only after careful preparation, if time permits Even when proper preparation is not possible the results have often been good When congestive failure exists rapid intravenous digitalization should be carried out and mercurial diuretics should be given In patients with rheumatic heart disease and auricular fibrillation the rate should be slowed by intravenous digitalis therapy before surgery is attempted Persons with compensated heart disease may be expected to

undergo operations as well as those whose hearts are normal if certain precautions are taken. Special attention should be paid to the amount of fluids and sodium given pre and postoperatively and an adequate quantity of oxygen should be administered during the operation. The administration of large quantities of intravenous saline after an operative procedure should be avoided in cardiac patients. The measures suggested reduce the incidence of pulmonary edema and congestive heart failure.

In patients with coronary artery disease particular care should be exercised to avoid anoxemia especially during the induction of anesthesia. The most desirable anesthetic of course, is a local or regional one. Spinal anesthesia has usually been considered dangerous for patients with coronary artery disease or severe hypertension but we have found it effective and safe for operations on the lower abdomen or pelvis. Intramuscular neosynephrine or ephedrine may be given prophylactically to prevent a precipitous fall in blood pressure during spinal anesthesia and intravenous neosynephrine (0.5 to 1 mg.) should be available for immediate use if this should occur.

If a general anesthetic is used ether is preferred or a combination of pentothal oxygen and cyclopropane may be used. Since the latter may induce ventricular arrhythmias[131] quinidine two doses of 6 gr (4 gm) orally, Pronestyl[134] (1 to 2 gm.) or Dibenamine (7 to 7.5 mg./kg.)[199] should be given before the operation particularly in patients with a history of arrhythmias. These agents markedly reduce the incidence of tachycardias that occur during surgical procedures. If auricular tachycardia flutter or fibrillation occurs quinidine should be given intramuscularly. If a ventricular arrhythmia sets in despite these prophylactic measures Pronestyl should be injected intravenously.

The incidence of postoperative thromboembolism and pulmonary infarction is higher in cardiac than in noncardiac patients. The single most important factor in preventing these *complications is early ambulation, par*

ticularly in the obese The patient should also be instructed to turn in bed as soon as possible to move his legs within several hours after operation, and to avoid straining at stool If pulmonary infarction occurs, despite these measures, treatment is given as outlined in Chapter 3 p 83

Cardiac arrest may occur during the course of any operative procedure in both normal and cardiac patients It may be due to marked vagal stimulation during the operation and can be prevented in some cases by the intravenous administration of atropine, 0 6 mg (1/100 gr), sixty minutes before surgery, or 0 3 mg (1/50 gr) intramuscularly thirty minutes before[132] Cardiac arrest occurs in operations on the chest more often than in abdominal or pelvic surgery When it does occur, the instantaneous execution of a well planned course of treatment is essential to save the patient's life

A sixteen year old boy, who was operated upon for atelectasis of the left lung secondary to fibrosarcoma of the bronchus, furnishes a dramatic example of the success of cardiac resuscitation, even after a forty minute period of cardiac arrest * He received cyclopropane and ether at 8 10 A M, at 8 25 A M, an oval endotracheal tube was introduced At 8 27, the left hemithorax was entered Sixty mg of procaine HCL was injected into the tubing of an intravenous drip The pulmonary artery was ligated at 9 05, when cardiac arrest occurred Artificial respiration was begun immediately by intermittent manual compression of the breathing bag of the anesthetic apparatus Simultaneously, manual massage of the heart was started at a rate of 50 per minute At 9 10, a mixture of 80 mg of procaine HCL and 1 mg epinephrine was injected into the left ventricular cavity At 9 25, 60 mg of procaine and 0 5 mg epinephrine were given and at 9 30, 80 mg more of procaine were injected Cardiac action did not return At 9 45, the pericardium was grasped with a hemostat,

* This case is described with the permission of the operating surgeon Dr Arthur S W Touroff[134]

preparatory to incision At that moment the heart suddenly beat violently and normal pulsation and respiration began The pneumonectomy was completed without mishap On the twelfth postoperative day the patient was entirely normal and has remained well

Restoration of heart action and maintenance of an adequate oxygen supply to the tissues are the two main objectives of therapy in these cases The first step is to expose the heart as quickly as possible preferably directly through the chest Sterile precautions are not required and the procedure may be carried out by anyone available Upon exposure of the heart cardiac arrest is usually seen but in some cases the collapse is the result of ventricular fibrillation If the latter is found 5 cc of a 1 per cent Procaine solution should be injected into the right auricle and the epicardial surface of the heart should be bathed by 2 to 3 cc [148] in preparation for defibrillation This is accomplished by placing electrodes on either side of the heart and passing a current of 110 volts and 1 5 amperes through it [9, 20] When cardiac standstill has thus been produced the treatment already described should be instituted Manual cardiac massage is started and continued at a steady rate of 50 to 80 per minute [30] until spontaneous cardiac action begins If there is no response 0 5 to 1 cc of a 1 1000 solution of epinephrine is injected into the right auricle [75] If the arrest still persists the massage and epinephrine injections should be continued for at least forty to sixty minutes before admitting failure When the heart is unusually flabby or atonic 3 to 5 cc of a 1 per cent solution of calcium chloride may be effective

Artificial respiration should be started simultaneously with the exposure of the heart if respirations have ceased If possible an intratracheal tube should be inserted and the lungs inflated and deflated by mechanical means This is by far the most efficient method of supplying oxygen [49] If endotracheal tubes are not available the resumption of respiration may be effected by mouth to mouth breathing

alternating manual pressure and release of the costal cage or the use of a tight fitting face mask pressure being applied intermittently When the latter method is used there is a real danger of forcing air into the stomach and causing marked gastric dilatation [273] One hundred per cent oxygen should be given and CO_2 mixtures should be avoided since the patient already has accumulated an excessive amount of CO_2 in the tissues Adequate oxygenation of the brain and other organs may be maintained by use of any one of the above procedures until spontaneous respiration and circulation are resumed

When hemorrhage has occurred and the blood volume is markedly decreased intra arterial transfusions of blood should be given at the rate of 150 to 200 cc minute These transfusions are given at a pressure higher than the systolic blood pressure

In many cases patients who had apparently expired will be completely revived, and will recover with no ill effects if the suggested treatment is undertaken promptly If the surgeon is properly equipped and psychologically ready for this emergency, good results may be expected

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